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INCA MUMMIES



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FEATURES

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BY IRA BLOCK

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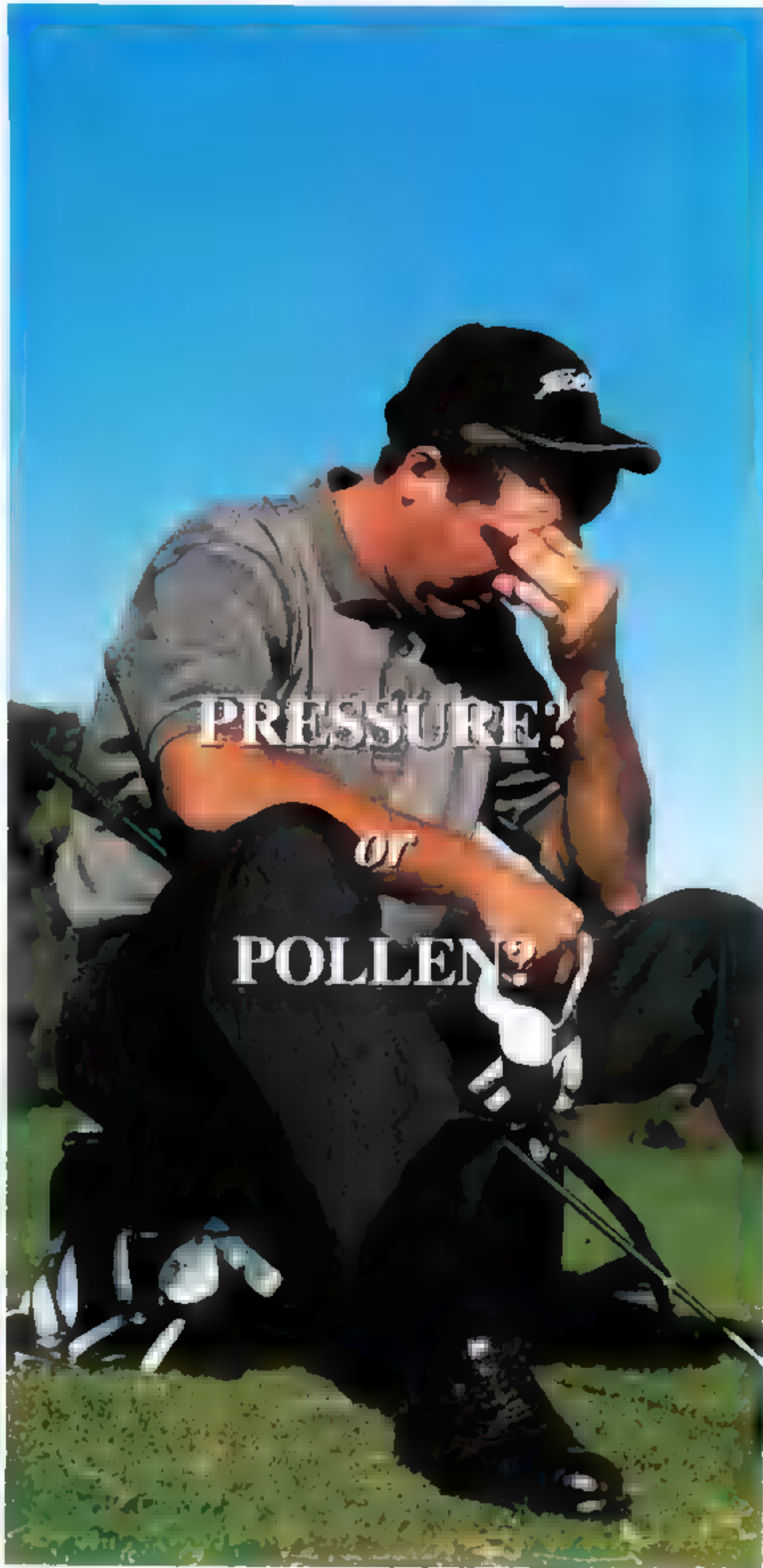
JIM RICHARDSON

The old expression “you are what you eat” raises a few more questions than it used to. It’s not as easy to know what’s in our food these days as it was back when much of it came from local family farms. Today huge corporations grow and distribute most of what we eat, often using assembly-line techniques like those pictured above at an Arkansas chicken processor. Large-scale production has lowered the cost of foods, but it also poses risks. That chicken salad might be tinged with *Salmonella*. That juicy burger could harbor deadly *E. coli*. Even vegetarians aren’t safe: Government agencies warn that sprouts are really only safely consumed when cooked. Producers often dose our food animals with antibiotics—some of the same ones we use to treat human illnesses—to make them grow bigger and get them to market faster. Can we afford the kinds of antibiotic resistance that might result?

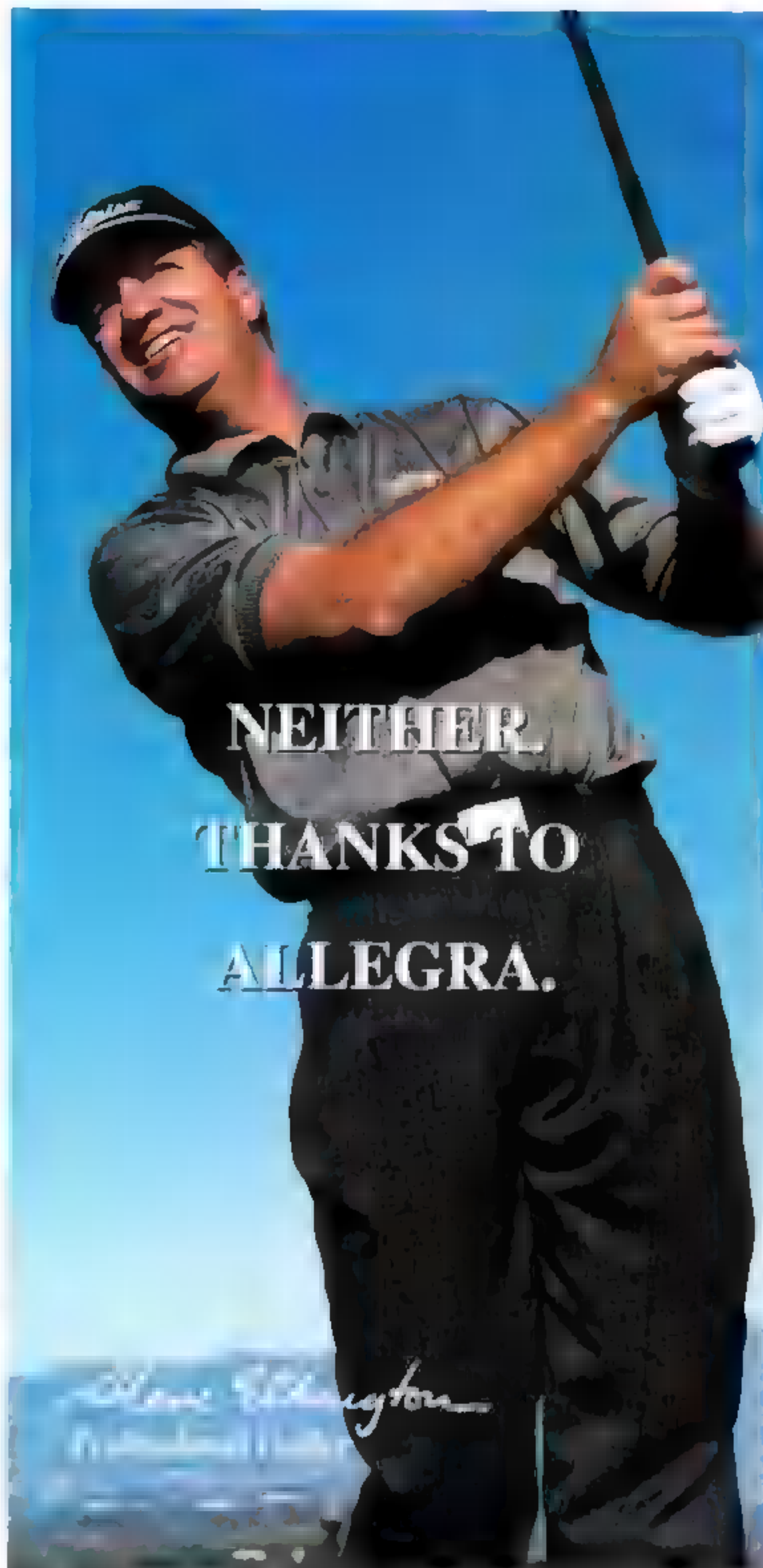
Genetic engineers are a big part of the food picture too. Novel methods for enhancing the genetic traits of our food crops raise hopes of feeding the world. But some people wonder if we know the answers to all questions about how biotech foods will affect the environment and us.

Jennifer Ackerman and Jim Richardson examine these questions in two stories in this issue. Take a bite. It’s good for you.

Bill Allen



**PRESSURE?
OR
POLLEN?**



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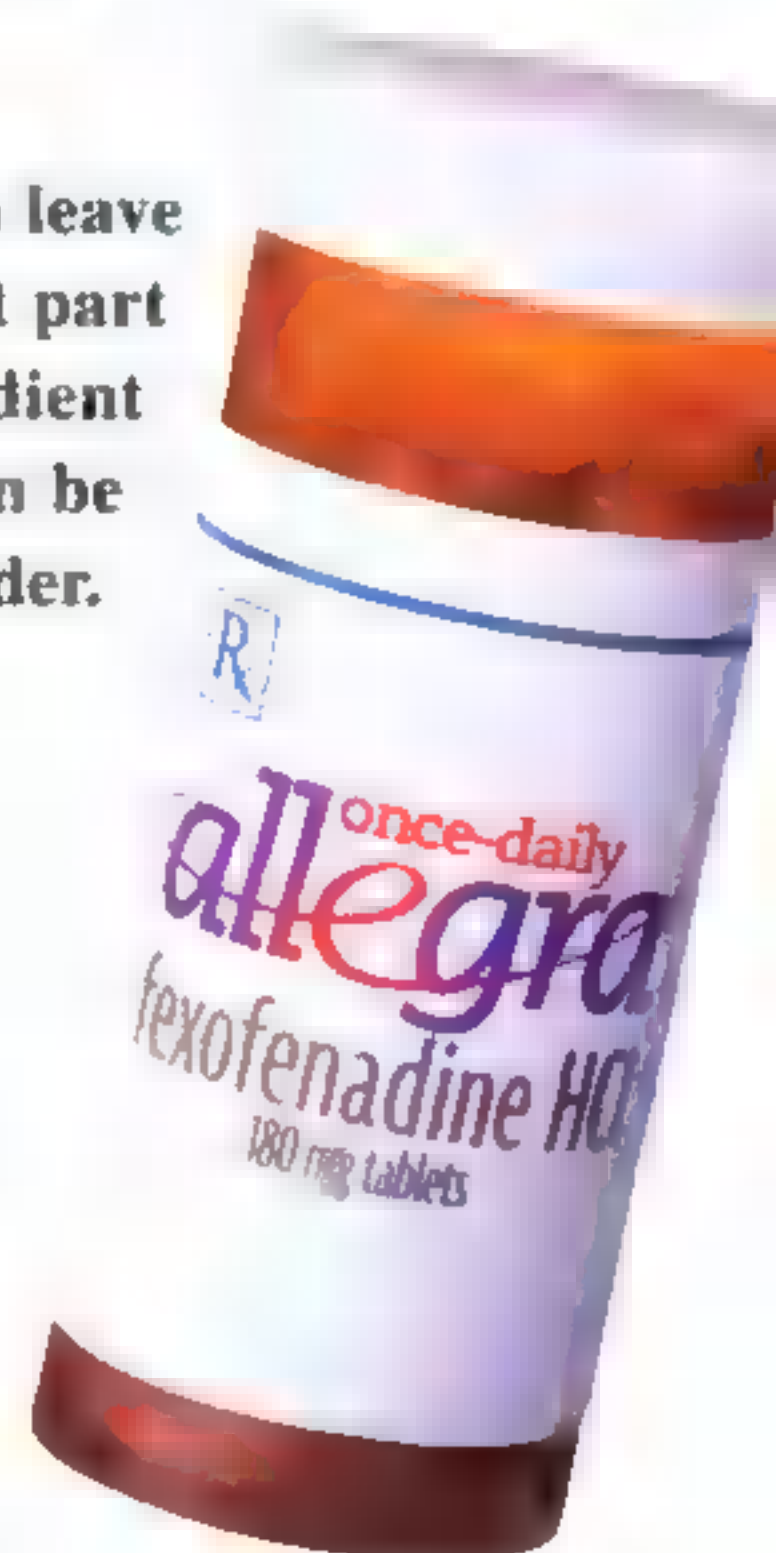
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INDICATIONS AND USAGE

Seasonal Allergic Rhinitis

ALLEGRA is indicated for the relief of symptoms associated with seasonal allergic rhinitis in adults and children 6 years of age and older. Symptoms treated effectively were sneezing, rhinorrhea, itchy nose/palate/throat, itchy/watery/ired eyes.

Chronic Idiopathic Urticaria

ALLEGRA is indicated for treatment of uncomplicated skin manifestations of chronic idiopathic urticaria in adults and children 6 years of age and older. It significantly reduces pruritus and the number of wheals.

CONTRAINDICATIONS

ALLEGRA is contraindicated in patients with known hypersensitivity to any of its ingredients.

PRECAUTIONS

Drug Interaction with Erythromycin and Ketoconazole

Fexofenadine hydrochloride has been shown to exhibit minimal (ca. 5%) metabolism. However, co-administration of fexofenadine hydrochloride with ketoconazole and erythromycin led to increased plasma levels of fexofenadine hydrochloride. Fexofenadine hydrochloride had no effect on the pharmacokinetics of erythromycin and ketoconazole. In two separate studies, fexofenadine hydrochloride 120 mg twice daily (two times the recommended twice daily dose) was co-administered with erythromycin 500 mg every 6 hours or ketoconazole 400 mg once daily under steady-state conditions to normal, healthy volunteers (n=24, each study). No differences in adverse events or QT_c interval were observed when patients were administered fexofenadine hydrochloride alone or in combination with erythromycin or ketoconazole. The findings of these studies are summarized in the following table:

Effects on steady-state fexofenadine hydrochloride pharmacokinetics after 7 days of co-administration with fexofenadine hydrochloride 120 mg every 12 hours (two times the recommended twice daily dose) in normal volunteers (n=24)

Concomitant Drug	C _{max,SS} (Peak plasma concentration)	AUC _{0-12h} (Extent of systemic exposure)
Erythromycin (500 mg every 6 hrs)	+82%	+109%
Ketoconazole (400 mg once daily)	+135%	+154%

The changes in plasma levels were within the range of plasma levels achieved in adequate and well-controlled clinical trials.

The mechanism of these interactions has been evaluated in *in vitro*, *in situ*, and *in vivo* animal models. These studies indicate that ketoconazole or erythromycin co-administration enhances fexofenadine gastrointestinal absorption. *In vivo* animal studies also suggest that in addition to increasing absorption, ketoconazole decreases fexofenadine hydrochloride gastrointestinal secretion, while erythromycin may also decrease biliary excretion.

Drug Interactions with Antacids

Administration of 120 mg of fexofenadine hydrochloride (2 x 60 mg capsule) within 15 minutes of an aluminum and magnesium containing antacid (Maalox[®]) decreased fexofenadine AUC by 41% and C_{max} by 43%. ALLEGRA should not be taken closely in time with aluminum and magnesium containing antacids.

Carcinogenesis, Mutagenesis, Impairment of Fertility

The carcinogenic potential and reproductive toxicity of fexofenadine hydrochloride were assessed using terfenadine studies with adequate fexofenadine hydrochloride exposure (based on plasma area-under-the-concentration vs. time [AUC] values). No evidence of carcinogenicity was observed in an 18-month study in mice and in a 24-month study in rats at oral doses up to 150 mg/kg of terfenadine (which led to fexofenadine exposures that were respectively approximately 3 and 5 times the exposure from the maximum recommended daily oral dose of fexofenadine hydrochloride in adults and children).

In vitro (Bacterial Reverse Mutation, CHO/HGPRT Forward Mutation, and Rat Lymphocyte Chromosomal Aberration assays) and *in vivo* (Mouse Bone Marrow Micronucleus assay) tests, fexofenadine hydrochloride revealed no evidence of mutagenicity.

In rat fertility studies, dose-related reductions in implants and increases in postimplantation losses were observed at an oral dose of 150 mg/kg of terfenadine (which led to fexofenadine hydrochloride exposures that were approximately 3 times the exposure of the maximum recommended daily oral dose of fexofenadine hydrochloride in adults).

Pregnancy

Teratogenic Effects: Category C. There was no evidence of teratogenicity in rats or rabbits at oral doses of terfenadine up to 300 mg/kg (which led to fexofenadine exposures that were approximately 4 and 31 times, respectively, the exposure from the maximum recommended daily oral dose of fexofenadine in adults).

There are no adequate and well-controlled studies in pregnant women. Fexofenadine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nonteratogenic Effects. Dose-related decreases in pup weight gain and survival were observed in rats exposed to an oral dose of 150 mg/kg of terfenadine (approximately 3 times the maximum recommended daily oral dose of fexofenadine hydrochloride in adults based on comparison of fexofenadine hydrochloride AUCs).

Nursing Mothers

There are no adequate and well-controlled studies in women during lactation. Because many drugs are excreted in human milk, caution should be exercised when fexofenadine hydrochloride is administered to a nursing woman.

Pediatric Use

The recommended dose in patients 6 to 11 years of age is based on cross-study comparison of the pharmacokinetics of ALLEGRA in adults and pediatric patients and on the safety profile of fexofenadine hydrochloride in both adult and pediatric patients at doses equal to or higher than the recommended doses.

The safety of ALLEGRA tablets at a dose of 30 mg twice daily has been demonstrated in 438 pediatric patients 6 to 11 years of age in two placebo-controlled 2-week seasonal allergic rhinitis trials. The safety of ALLEGRA for the treatment of chronic idiopathic urticaria in patients 6 to 11 years of age is based on cross-study comparison of the pharmacokinetics of ALLEGRA in adult and pediatric patients and on the safety profile of fexofenadine in both adult and pediatric patients at doses equal to or higher than the recommended dose.

The effectiveness of ALLEGRA for the treatment of seasonal allergic rhinitis in patients 6 to 11 years of age was demonstrated in one trial (n=411) in which ALLEGRA tablets 30 mg twice daily significantly reduced total symptom scores compared to placebo, along with extrapolation of demonstrated efficacy in patients ages 12 years and older, and the pharmacokinetic comparisons in adults and children. The effectiveness of ALLEGRA for the treatment of chronic idiopathic urticaria in patients 6 to 11 years of age is based on an extrapolation of the demonstrated efficacy of ALLEGRA in adults with this condition and the likelihood that the disease course, pathophysiology and the drug's effect are substantially similar in children to that of adult patients. The safety and effectiveness of ALLEGRA in pediatric patients under 6 years of age have not been established.

Geriatric Use

Clinical studies of ALLEGRA tablets and capsules did not include sufficient numbers of subjects aged 65 years and over to determine whether this population responds differently from younger patients. Other reported clinical experience has not identified differences in responses between the geriatric and younger patients. This drug is known to be substantially excreted by the kidney, and the risk of toxic reactions to this drug may be greater in patients with impaired renal function. Because elderly patients are more likely to have decreased renal function, care should be taken in dose selection, and may be useful to monitor renal function. (See CLINICAL PHARMACOLOGY).

ADVERSE REACTIONS

Seasonal Allergic Rhinitis

Adults. In placebo-controlled seasonal allergic rhinitis clinical trials in patients 12 years of age and older, which included 2461 patients receiving fexofenadine hydrochloride capsules at doses of 20 mg to 240 mg twice daily, adverse events were similar in fexofenadine hydrochloride and placebo-treated patients. All

adverse events that were reported by greater than 1% of patients who received the recommended daily dose of fexofenadine hydrochloride (60 mg capsules twice daily), and that were more common with fexofenadine hydrochloride than placebo, are listed in Table 1.

In a placebo-controlled clinical study in the United States, which included 570 patients aged 12 years and older receiving fexofenadine hydrochloride tablets at doses of 120 or 180 mg once daily, adverse events were similar in fexofenadine hydrochloride and placebo-treated patients. Table 1 also lists adverse experiences that were reported by greater than 2% of patients treated with fexofenadine hydrochloride tablets at doses of 180 mg once daily and that were more common with fexofenadine hydrochloride than placebo.

The incidence of adverse events, including drowsiness, was not dose-related and was similar across subgroups defined by age, gender, and race.

Table 1
Adverse experiences in patients ages 12 years and older reported in placebo-controlled seasonal allergic rhinitis clinical trials in the United States

Adverse experience	Twice daily dosing with fexofenadine capsules at rates of greater than 1%	
	Fexofenadine 60 mg Twice Daily (n=679)	Placebo Twice Daily (n=671)
Viral (cold, flu)	2.5%	1.5%
Nausea	1.6%	1.5%
Dysmenorrhea	1.5%	0.3%
Drowsiness	1.3%	0.9%
Dyspepsia	1.3%	0.6%
Fatigue	1.3%	0.9%

Once daily dosing with fexofenadine hydrochloride tablets at rates of greater than 2%

Adverse experience	Twice daily dosing with fexofenadine hydrochloride tablets at rates of greater than 2%	
	Fexofenadine 180 mg once daily (n=283)	Placebo (n=293)
Headache	10.6%	7.5%
Upper Respiratory Tract Infection	3.2%	3.1%
Muscle Pain	2.8%	1.4%

The frequency and magnitude of laboratory abnormalities were similar in fexofenadine hydrochloride and placebo-treated patients.

Pediatric. Table 2 lists adverse experiences in patients aged 6 to 11 years of age which were reported by greater than 2% of patients treated with fexofenadine hydrochloride tablets at a dose of 30 mg twice daily in placebo-controlled seasonal allergic rhinitis studies in the United States and Canada that were more common with fexofenadine hydrochloride than placebo.

Table 2
Adverse experiences reported in placebo-controlled seasonal allergic rhinitis studies in pediatric patients ages 6 to 11 in the United States and Canada at rates of greater than 2%

Adverse experience	Twice daily dosing with fexofenadine hydrochloride tablets at rates of greater than 2%	
	Fexofenadine 30 mg twice daily (n=209)	Placebo (n=229)
Headache	7.2%	6.6%
Accidental Injury	2.9%	1.3%
Coughing	3.8%	1.3%
Fever	2.4%	0.9%
Pain	2.4%	0.4%
Otitis Media	2.4%	0.0%
Upper Respiratory Tract Infection	4.3%	1.7%

Chronic Idiopathic Urticaria

Adverse events reported by patients 12 years of age and older in placebo-controlled chronic idiopathic urticaria studies were similar to those reported in placebo-controlled seasonal allergic rhinitis studies. In placebo-controlled chronic idiopathic urticaria clinical trials, which included 726 patients 12 years of age and older receiving fexofenadine hydrochloride tablets at doses of 20 to 240 mg twice daily, adverse events were similar in fexofenadine hydrochloride and placebo-treated patients. Table 3 lists adverse experiences in patients aged 12 years and older which were reported by greater than 2% of patients treated with fexofenadine hydrochloride 60 mg tablets twice daily in controlled clinical studies in the United States and Canada and that were more common with fexofenadine hydrochloride than placebo. The safety of fexofenadine hydrochloride in the treatment of chronic idiopathic urticaria in pediatric patients 6 to 11 years of age is based on the safety profile of fexofenadine hydrochloride in adults and adolescent patients at doses equal to or higher than the recommended dose (see Pediatric Use).

Table 3
Adverse experiences reported in patients 12 years and older in placebo-controlled chronic idiopathic urticaria studies in the United States and Canada at rates of greater than 2%

Adverse experience	Twice daily dosing with fexofenadine hydrochloride tablets at rates of greater than 2%	
	Fexofenadine 60 mg twice daily (n=186)	Placebo (n=178)
Back Pain	2.2%	1.1%
Sinusitis	2.2%	1.1%
Dizziness	2.2%	0.6%
Drowsiness	2.2%	0.0%

Events that have been reported during clinical trials involving seasonal allergic rhinitis and chronic idiopathic urticaria patients with incidences less than 1% and similar to placebo and have been rarely reported during postmarketing surveillance include: insomnia, nervousness, and sleep disorders or parosmia. In rare cases, rash, urticaria, pruritus and hypersensitivity reactions with manifestations such as angioedema, chest tightness, dyspnea, flushing and systemic anaphylaxis have been reported.

OVERDOSAGE

Reports of fexofenadine hydrochloride overdose have been infrequent and contain limited information. However, dizziness, drowsiness, and dry mouth have been reported. Single doses of fexofenadine hydrochloride up to 800 mg (six normal volunteers at this dose level), and doses up to 690 mg twice daily for 1 month (three normal volunteers at this dose level) or 240 mg once daily for 1 year (234 normal volunteers at this dose level) were administered without the development of clinically significant adverse events as compared to placebo.

In the event of overdose, consider standard measures to remove any unabsorbed drug. Symptomatic and supportive treatment is recommended.

Hemodialysis did not effectively remove fexofenadine hydrochloride from blood (1.7% removed) following terfenadine administration.

No deaths occurred at oral doses of fexofenadine hydrochloride up to 5000 mg/kg in mice (110 times the maximum recommended daily oral dose in adults and 200 times the maximum recommended daily oral dose in children based on mg/m²) and up to 5000 mg/kg in rats (230 times the maximum recommended daily oral dose in adults and 400 times the maximum recommended daily oral dose in children based on mg/m²). Additionally, no clinical signs of toxicity or gross pathological findings were observed. In dogs, no evidence of toxicity was observed at oral doses up to 2000 mg/kg (300 times the maximum recommended daily oral dose in adults and 530 times the maximum recommended daily oral dose in children based on mg/m²).

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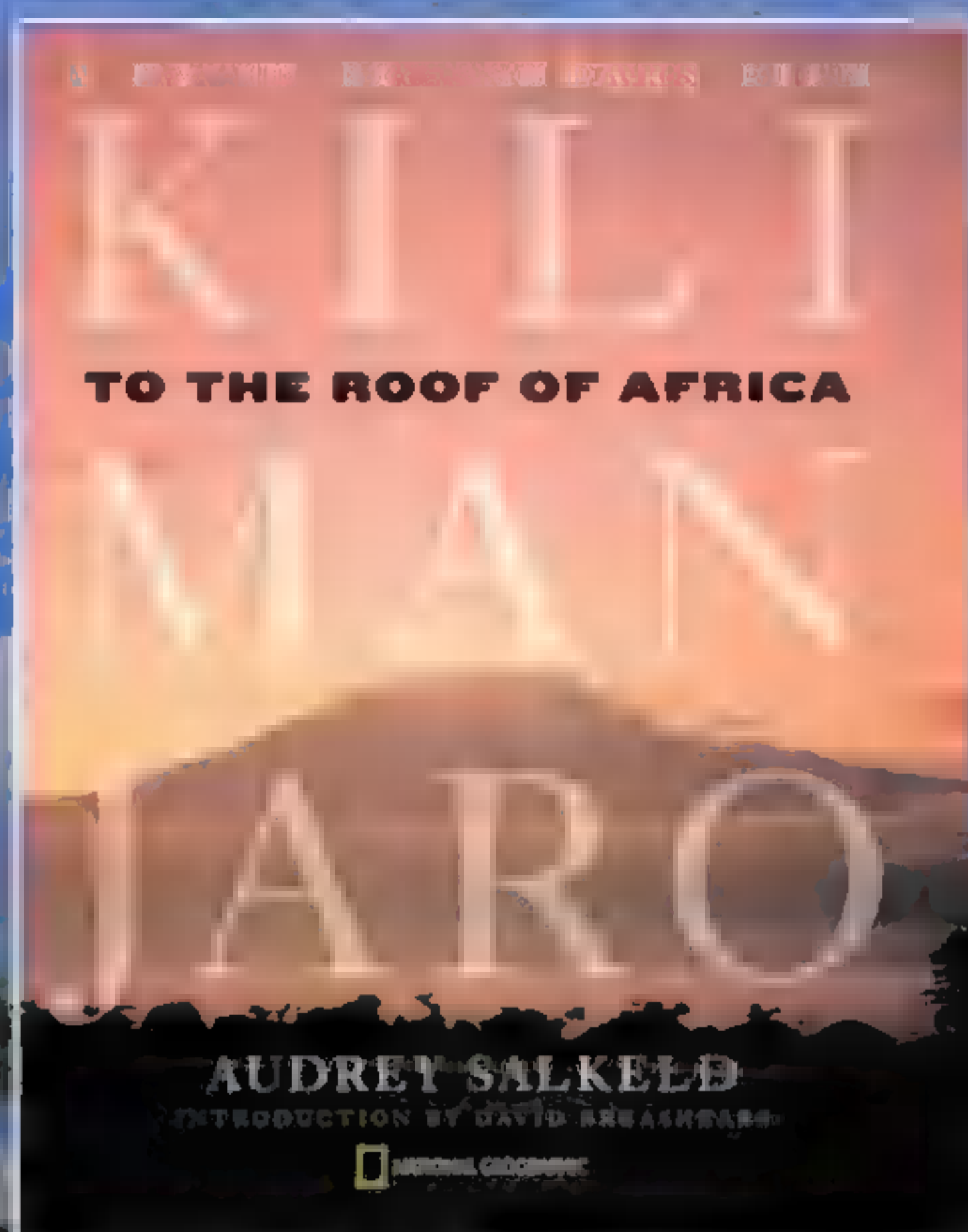
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Forum

January 2002

The debut of the euro marked a "New Europe," poised for peace and unity. For some, the new currency just means more tangled bureaucracy, but others see it as a promise of opportunity. A survivor of the horrors of WWII wrote: "We lost our home, but we had our lives. There will be obstacles in uniting Europe, but that is a small price to pay for lasting peace on the continent."



Wolf to Woof

I was struck by the timing of "Wolf to Woof." Here in Alaska the comment period was ending on proposals to continue the use of snowmobiles in hunting wolves. At its fastest a wolf cannot outrun a snowmobile. In such a match inevitably the wolf will either be run over and killed or run to exhaustion, then shot where it lies. As abhorrent as that practice may seem, it was permitted in certain areas of the state where reports from trappers seemed to indicate a great decline in moose caused by wolves. Data supplied by Alaska's Department of Fish and Game, however, disproves the claims.

ART GREENWALT
Fairbanks, Alaska

Can you explain the numbers on the photograph on page 10 titled "Dog of the Dead"?

STEPHEN J. RIGGI
Ipswich, Massachusetts

The number 6743 is a label stamped on the mummy by the

British Museum. "Salt 1821" refers to British Consul General Henry Salt, who acquired the artifact in 1821 while serving in Egypt. Salt sponsored several expeditions there and presented the British Museum with a colossal granite bust of Ramses II, among other artifacts.

Your article on the evolution of dogs was excellent. However, as a veterinary surgeon I was upset by the statement that "dogs are often used in experiments that might threaten human lives." Dogs tend to react like dogs and not like human beings, even when they're wired up in some laboratory. Two good examples are: A high level of the female hormone estrogen tends to delay blood clotting in female dogs while in women it has the opposite effect. Similarly, the injectable contraceptive Depo-Provera has been shown to cause mammary cancer in dogs but is apparently safe for women.

ANDRE MENACHE
London, England

World of Islam

I found "World of Islam" informative, but I was distressed by your characterizations of Christians and Muslims. When Muslims invaded, occupied,

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RICHARD OLSENIUS

Dogs—A Love Story

As makers of custom-built wheelchairs for disabled dogs, we hear every day from people like Ed Mulrenin in your article. While many of our clients at Eddie's Wheels for Pets are paraplegic due to hip dysplasia, neurological disease, and injury, an increasing number are simply old in body. We got into this business because

so many of us have shared the trauma of euthanizing an otherwise healthy dog when it ceased to be able to walk on its own.

LESLIE GRINNELL
Greenfield, Massachusetts

Pampering Tiffy with gourmet meals looks to me like an inversion of priorities. Pet owners who have more money than they know what to do with should consider donating it to charities, where it would actually make a difference.

DANIELLE TORIN
Williamstown, Massachusetts

I was at the World Trade Center assisting with the search-and-rescue efforts. During the first day the only moment that brought ■ smile to our faces

was watching a rescue dog play in the water. A golden retriever decided that he wanted to take a break and have some fun. With everything falling down around us, this dog let us know that eventually everything would be all right. We didn't hold on to each other, but we held on to that dog.

LT. SHAWN KUCHARSKI
Brooklyn, New York

I was disappointed that you barely mentioned one of the most ironic aspects of our love affair with dogs—millions of dogs are killed each year in shelters. People insist on buying purebreds while mutts die for want of a good home.

BARB JONES
*Charlottetown,
Prince Edward Island*

and subjugated North Africa, Spain, and the Middle East, you called them "an army empowered by faith." When Christians invaded, occupied, and subjugated a much smaller space, you said it was ■ "blood-soaked legacy—the indiscriminate killing of thousands of innocent Arabs." It is now politically correct to go easy on Muslims. When will it be politically correct to go easy on Christians? The truth is that all people fall short of their religious ideals, and some abuse religion for their own purposes.

STEVE SOUZA
Oregon House, California

I felt compelled to write to you after reading the quotation from Jennifer Calvo, a Catholic who converted to Islam. Western culture may be anathema to Muslims because of its superficiality in emphasizing money, youth, and sex appeal. This "crazy culture," however, isn't a result of Christianity's, Judaism's, or any religion's inability to provide a foundation of spirituality, morals, values, dress, and faith. One is empty if one chooses to be empty; one can find peace and spirituality in one's faith. One just has to look for it and want it.

AUSRA TALLAT KELPSA
Chicago, Illinois

professionals who do not have a well-connected relative have to migrate overseas to get a decent job. The private sector is even worse. Many qualified people in their 30s have never worked more than six months in a row in their whole lives. Mass media refers to us as the "lost generation." I am a translator who has mastered six languages, and the "postcard" Europe in your article has only given me the chance to be ■ bricklayer, postman, receptionist, and tow truck driver. Only the U.S. and Canada have given me a chance that my own continent denies me.

JOSÉ DELGADO
Madrid, Spain

WRITE TO FORUM

National Geographic Magazine, PO Box 98199, Washington, DC 20090-8199, or by fax to 202-828-5460, or via the Internet to ngsforum@nationalgeographic.com. Include name, address, and daytime telephone. Letters may be edited for clarity and space.

The New Europe

Although every fact stated in your article is true, I cannot see my daily reality reflected in it. Corruption and nepotism in the European Parliament and governmental agencies reach to such outrageous degrees that qualified

It will be really interesting to see how the EU fares economically. Finally there will be a group and currency that can challenge the U.S. economy and dollar. With a maximum of 550 million people in the EU and a strong currency,



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could the U.S. economy possibly take a backseat as number two? This might force it to move closer to Mexico and Canada, forming a 400-million-person-strong North American alliance using a common currency.

ANTHONY COZZI
Toronto, Ontario

If the European Union is so wonderful, why did it have to hold a summit behind barbed wire? Why were two recent EU referendums defeated by the people whose countries would be affected? Why did nearly 80 percent of the Swiss voting in a referendum last year reject the opportunity to even discuss joining the EU? The European Union is designed for politicians and bureaucrats and rejected by the people it claims to govern.

STEVE DOOLEY
Kingsbridge, England

I for one do not believe that Europe is ready for complete integration. I lack faith in a controlling entity run by a group of unelected commissioners and a parliament that cannot initiate primary legislation or even decide where to meet (it commutes, complete with baggage and interpreters, between Brussels and Strasbourg). I supported the Common Market and the European Community, but I fear a United States of Europe is a step too far and much too soon.

ALASDAIR COOK
Dumfries, Scotland

For years I have been observing negative reporting on Germany in the Anglo-Saxon press. Even this article went in that direction. The reports on almost all the nations in Europe were positive, but in Germany you found only neo-Nazis. At the last

If the European Union is so wonderful, why did it have to hold a summit behind barbed wire? . . . [The EU] is designed for politicians and bureaucrats and rejected by the people it claims to govern.

election, in 1998, the National Democratic Party of Germany received just 0.3 percent of the votes. All together the three right-wing parties have little more than 3 percent, much less than most European states.

KLAUS J. SCHWAND
Bad Camberg, Germany

In response to your statement in the caption on page 47 that Hungary's Roma are seeking protection from persecution, kindly note that last year Lungo Drom, a major Gypsy cultural and political association, formed an electoral alliance with Hungary's governing Young Democrats. This is the first time since the democratic changes in 1990 that a major political party has actively sought the involvement of the Gypsy-Roma community. Persecution? Hardly.

ILDIKO J. BODONI
Western Springs, Illinois

China's Unknown Gobi

Two photos in the January issue illustrate something I've never been able to understand. In the photo on page 59 of the Gobi story, a Chinese woman is using

a solar collector to cook. Then on page 106 of the Western Ghats article, a village woman near Nagarhole National Park in India carries a huge bundle of wood to fuel cooking fires. Why don't relief organizations put more effort into distributing appliances such as solar collectors and fuel-efficient ceramic stoves or teaching methods of drip irrigation and supplying the means and training to use them?

BETTY M. WALL
Hartville, Ohio

Conservation Hotspots

When reading E. O. Wilson's introduction, I was concerned to find the sentence "it should be possible to accommodate the continuing human surge while protecting a large part of Earth's threatened fauna and flora." The increasing population is the main problem, and it should not be "accommodated." Solutions should be sought to solve this problem, which will help solve many other problems including habitat destruction.

MICHAEL JUNKIN
Blacksburg, Virginia

ZipUSA: U.S.S. Enterprise

I recall encountering the *Enterprise* in 1962 while in the Navy. I was a radar operator, and I clocked it while it was doing its speed run. All I can say is that even with its immense size, it can outrun destroyers whose flank speed generally is around 35-40 knots. I can't tell you what we clocked it at because I'd probably be killed.

ALEX HOLUB
Los Angeles, California

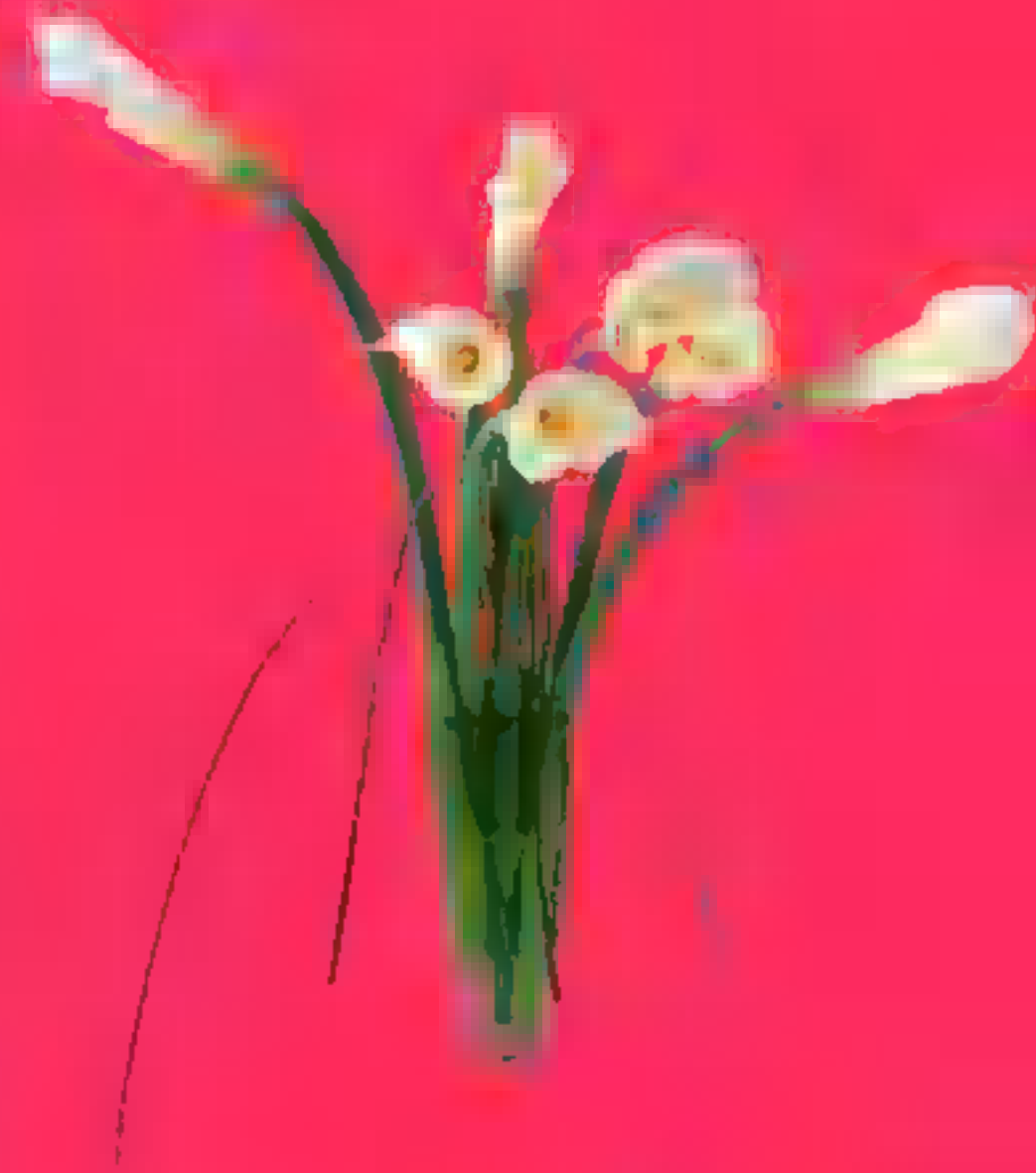
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A GLOBAL BURDEN

The Land Mine Wars

Demining efforts gain ground, but the threat remains

Kabir, a shepherd, was moving his flock when he became one more victim of Afghanistan's hidden terror. The land mine he stepped on, lurking a few inches deep in the earth, blew away his right leg and damaged his spine.

"Life will be very difficult for him," said Dr. Alberto Cairo, who cared for Kabir at a Kabul rehabilitation center funded by the International Committee of the Red Cross. Dr. Cairo has seen thousands of mine victims. "An accident puts a very big burden on the family," he said. "And Kabir has four children."

Watchdog groups estimate that every month 150 to 300 Afghans are killed or maimed by explosives, which include antitank and antipersonnel

mines as well as unexploded ordnance—UXO—such as grenades and artillery shells. About 20 percent of those treated in Afghan hospitals are children, like this boy (below, at right) who picked up a mine and lost a hand.

Employed in dozens of conflicts since coming into wide use in World War II, land mines keep on terrorizing; mines laid in Egypt in WWII are still known to cripple Bedouin nomads. Last year explosives killed or maimed 15,000 to 20,000 people in some 70 countries. Most were rural folk, like Zarko Peric (third page) who walks on a prosthesis, tagging along as his brother plows the family's plot in Bosnia and Herzegovina.

Afghanistan, after 23 years of



PAUL HANSEN

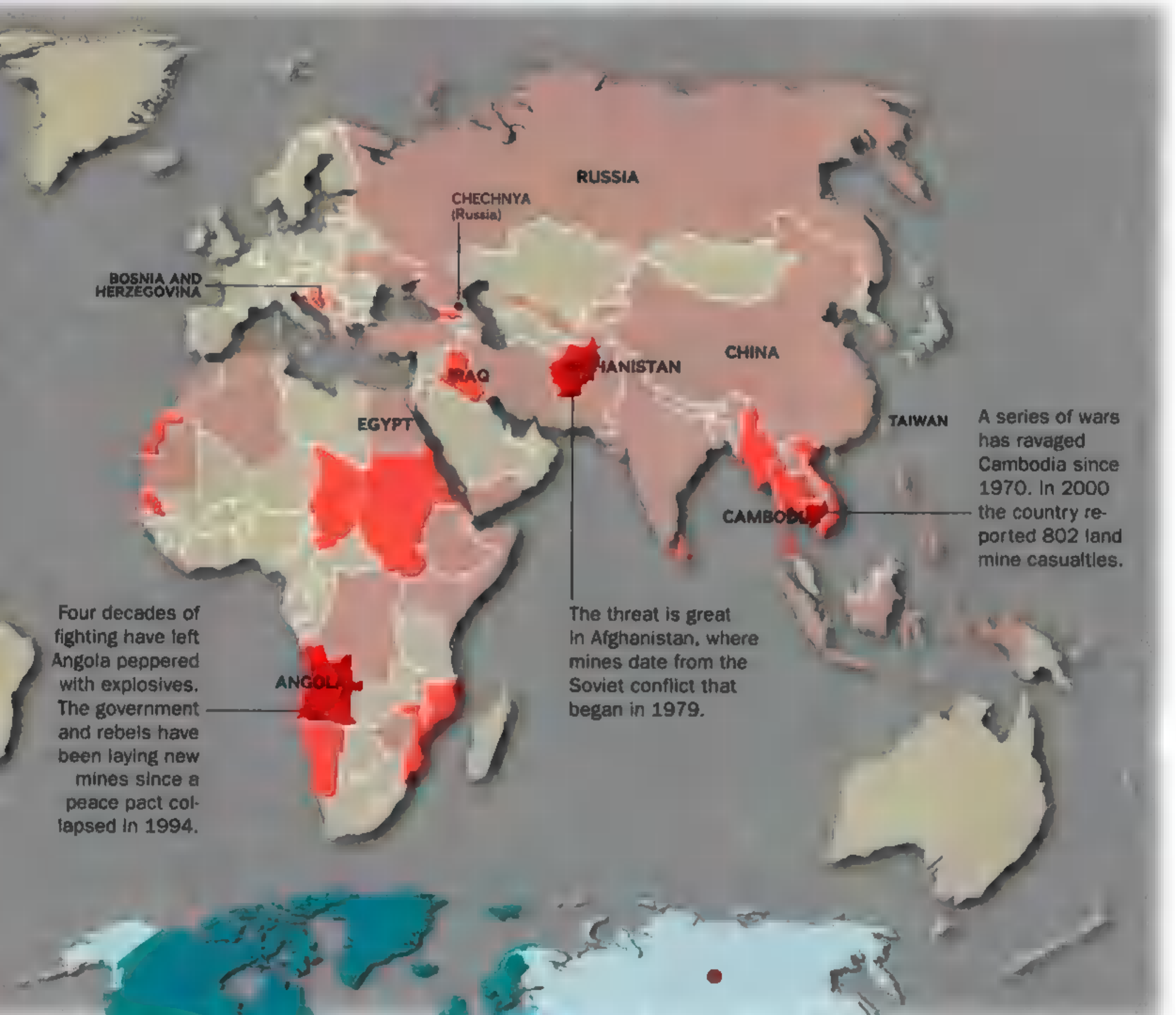


war, is probably the most mine-afflicted nation in the world. "I believe the worst are Afghanistan, Cambodia, and Angola, in that order," said deminer Paul Heslop of the Halo Trust, a British humanitarian organization.

The United Nations' Mine Action Program for Afghanistan—demining, marking mined areas, teaching civilians to be careful—employs nearly 5,000 Afghans, who earn \$100 to \$150 a month, princely wages

APHICA

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Banning the Mines

Land mine treaty

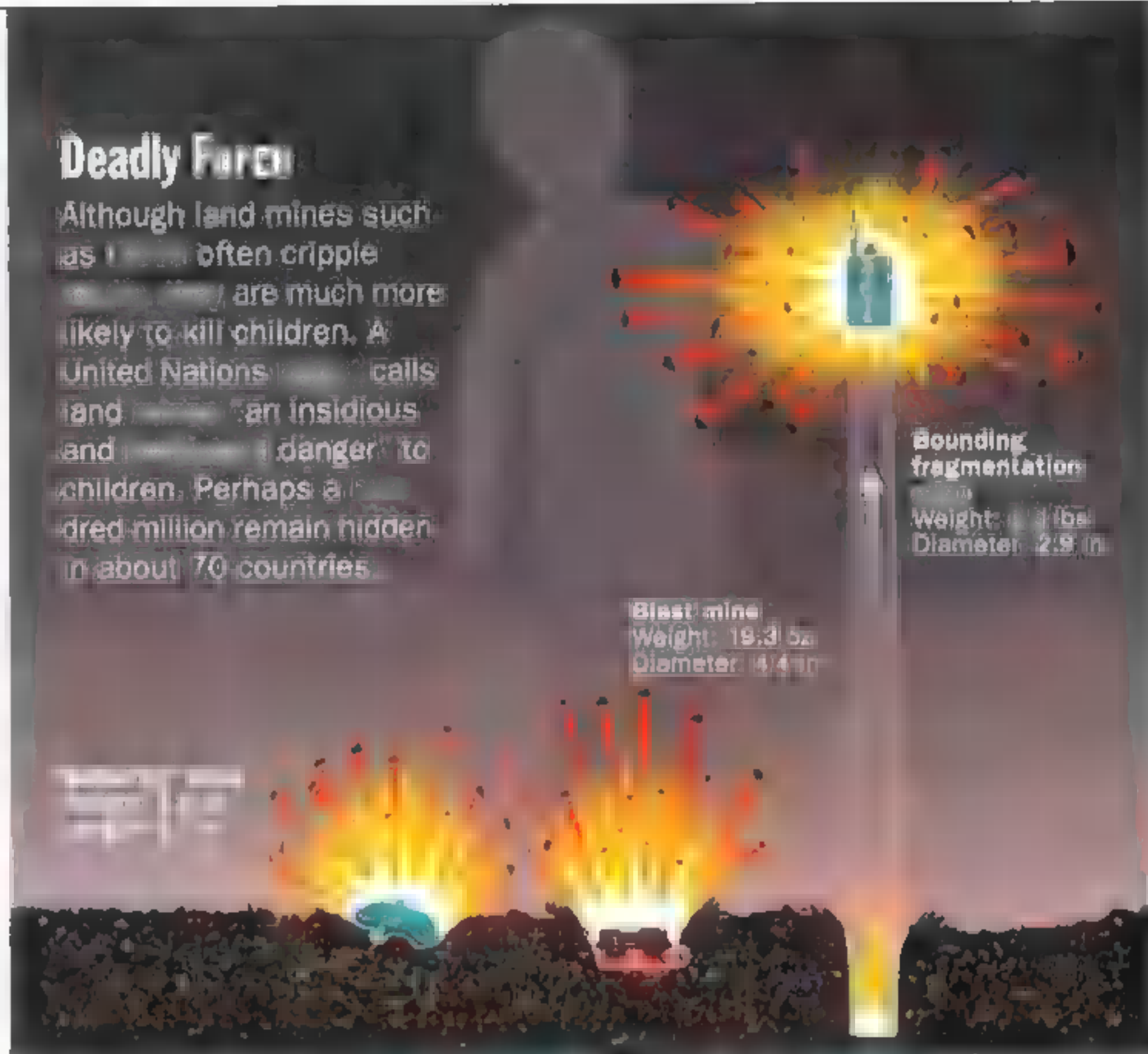
- Not signed
- Signed
- Signed and ratified
- Land mine producer

in their shattered homeland. Some follow dogs trained to detect mines by scent; when a dog gets a whiff of an explosive, he sits, awaiting his handler. Other Afghans drive bulldozers and cranes that clear minefields.

But the deminer equipped with a metal detector represents "our most important effort," said Heslop. "It's a dirty, dangerous job, no doubt about it. If you're stupid, you'll get killed or injured. But if you're careful and follow the rules, it's pretty straightforward." Rule No. 1: Don your armor, a Kevlar body protector and Plexiglas visor like those worn by a deminer in the Kurdish area of Iraq (below).



MICHAEL YAMASHITA (ABOVE), GUN DOLL



ART BY WILLIAM H. BOND

The most common mine in Afghanistan is about the size of a tuna fish can (art above, at center) and may be detonated by a footfall. Another pops up and explodes waist high, spewing steel wires (art, at right). During its Afghan invasion, 1979-1989, the Soviet Union salted vast areas with "butterfly" mines (art, at left), often dropped from helicopters. Plastic wings eased their descent.

Mounting an aggressive

campaign, humanitarian organizations such as the International Campaign to Ban Land Mines have persuaded more than 140 nations to commit to a ban of antipersonnel mines. The U.S., Russia, and China have yet to sign on.

Russia (in Chechnya) also numbers among the war-torn countries where mines continue to be deployed. But more mines are being cleared than laid worldwide, and casualty figures, while ghastly, are slowly declining. These achievements have been wrought at a cost of more than a billion dollars, almost all donated by governments, including 300 million from the U.S.

In Afghanistan deminers have cleared mines and UXO from a total of 86 square miles of roads, towns, and villages, as well as from 124 square miles of battlefield. But to finally rid the country of its explosives will take at least ten more years—and cost another half billion dollars.



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LIPITOR® (atorvastatin calcium) is a prescription drug used with diet to lower cholesterol. LIPITOR is not for everyone, including those with liver disease or possible liver problems, women who are nursing, pregnant, or may become pregnant. LIPITOR has not been shown to prevent heart disease or heart attacks.

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Please see additional important information on next page.

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Brief Summary of Prescribing Information

CONTRAINDICATIONS: Active liver disease or unexplained persistent elevations of serum transaminases. Hypersensitivity to any component of this medication. **Pregnancy and Lactation** — Atherosclerosis is a chronic process and discontinuation of lipid-lowering drugs during pregnancy should have little impact on the outcome of long-term therapy of primary hypercholesterolemia. Cholesterol and other products of cholesterol biosynthesis are essential components for fetal development (including synthesis of steroids and cell membranes). Since HMG-CoA reductase inhibitors decrease cholesterol synthesis and possibly the synthesis of other biologically active substances derived from cholesterol, they may cause fetal harm when administered to pregnant women. Therefore, HMG-CoA reductase inhibitors are contraindicated during pregnancy and in nursing mothers. **ATORVASTATIN SHOULD BE ADMINISTERED TO WOMEN OF CHILD-BEARING AGE ONLY WHEN SUCH PATIENTS ARE HIGHLY UNLIKELY TO CONCEIVE AND HAVE BEEN INFORMED OF THE POTENTIAL HAZARDS.** If the patient becomes pregnant while taking this drug, therapy should be discontinued and the patient apprised of the potential hazard to the fetus.

WARNINGS: Liver Dysfunction — HMG-CoA reductase inhibitors, like some other lipid-lowering therapies, have been associated with biochemical abnormalities of liver function. **Persistent elevations (>3 times the upper limit of normal [ULN]) occurring on 2 or more occasions) in serum transaminases occurred in 0.7% of patients who received atorvastatin in clinical trials. The incidence of these abnormalities was 0.2%, 0.2%, 0.6%, and 2.3% for 10, 20, 40, and 80 mg, respectively.** One patient in clinical trials developed jaundice. Increases in liver function tests (LFT) in other patients were not associated with jaundice or other clinical signs or symptoms. Upon dose reduction, drug interruption, or discontinuation, transaminase levels returned to or near pretreatment levels without sequelae. Eighteen of 30 patients with persistent LFT elevations continued treatment with a reduced dose of atorvastatin. It is recommended that liver function tests be performed prior to and at 12 weeks following both the initiation of therapy and any elevation of dose, and periodically (eg, semiannually) thereafter. Liver enzyme changes generally occur in the first 3 months of treatment with atorvastatin. Patients who develop increased transaminase levels should be monitored until the abnormalities resolve. Should an increase in ALT or AST of >3 times ULN persist, reduction of dose or withdrawal of atorvastatin is recommended. Atorvastatin should be used with caution in patients who consume substantial quantities of alcohol and/or have a history of liver disease. Active liver disease or unexplained persistent transaminase elevations are contraindications to the use of atorvastatin (see CONTRAINDICATIONS). **Skeletal Muscle** — **Rhabdomyolysis with acute renal failure secondary to myoglobinuria has been reported with other drugs in this class.** Uncomplicated myalgia has been reported in atorvastatin-treated patients (see ADVERSE REACTIONS). Myopathy, defined as muscle aches or muscle weakness in conjunction with increases in creatine phosphokinase (CPK) values >10 times ULN, should be considered in any patient with diffuse myalgias, muscle tenderness or weakness, and/or marked elevation of CPK. Patients should be advised to report promptly unexplained muscle pain, tenderness or weakness, particularly if accompanied by malaise or fever. Atorvastatin therapy should be discontinued if markedly elevated CPK levels occur or myopathy is diagnosed or suspected. The risk of myopathy during treatment with other drugs in this class is increased with concurrent administration of cyclosporine, fibric acid derivatives, erythromycin, niacin, or azole antifungals. Physicians considering combined therapy with atorvastatin and fibric acid derivatives, erythromycin, immunosuppressive drugs, azole antifungals, or lipid-lowering doses of niacin should carefully weigh the potential benefits and risks and should carefully monitor patients for any signs or symptoms of muscle pain, tenderness, or weakness, particularly during the initial months of therapy and during any periods of upward dosage titration of either drug. Periodic creatine phosphokinase (CPK) determinations may be considered in such situations, but there is no assurance that such monitoring will prevent the occurrence of severe myopathy. **Atorvastatin therapy should be temporarily withheld or discontinued in any patient with an acute, serious condition suggestive of a myopathy or having a risk factor predisposing to the development of renal failure secondary to rhabdomyolysis (eg, severe acute infection, hypotension, major surgery, trauma, severe metabolic, endocrine and electrolyte disorders, and uncontrolled seizures).**

PRECAUTIONS: General — Before instituting therapy with atorvastatin, an attempt should be made to control hypercholesterolemia with appropriate diet, exercise, and weight reduction in obese patients, and to treat other underlying medical problems (see INDICATIONS AND USAGE in full prescribing information). **Information for Patients** — Patients should be advised to report promptly unexplained muscle pain, tenderness, or weakness, particularly if accompanied by malaise or fever. **Drug Interactions** — The risk of myopathy during treatment with other drugs of this class is increased with concurrent administration of cyclosporine, fibric acid derivatives, niacin (nicotinic acid), erythromycin, azole antifungals (see WARNINGS, Skeletal Muscle). **Anticid**: When atorvastatin and Meolox® TC suspension were coadministered, plasma concentrations of atorvastatin decreased approximately 35%. However, LDL-C reduction was not altered. **Antipyrene**: Because atorvastatin does not affect the pharmacokinetics of antipyrene, interactions with other drugs metabolized via the same cytochrome isozymes are not expected. **Colestipol**: Plasma concentrations of atorvastatin decreased approximately 25% when colestipol and atorvastatin were coadministered. However, LDL-C reduction was greater when atorvastatin and colestipol were coadministered than when either drug was given alone. **Cimetidine**: Atorvastatin plasma concentrations and LDL-C reduction were not altered by coadministration of cimetidine. **Digoxin**: When multiple doses of atorvastatin and digoxin were coadministered, steady state plasma digoxin concentrations increased by approximately 20%. Patients taking digoxin should be monitored appropriately. **Erythromycin**: In healthy individuals, plasma concentrations of atorvastatin increased approximately 40% with coadministration of atorvastatin and erythromycin, a known inhibitor of cytochrome P450 3A4 (see WARNINGS, Skeletal Muscle). **Oral Contraceptives**: Coadministration of atorvastatin and an oral contraceptive increased AUC values for norethindrone and ethinyl estradiol by approximately 30% and 20%, respectively. These increases should be considered when selecting an oral contraceptive for a woman taking atorvastatin. **Warfarin**: Atorvastatin had no clinically significant effect on prothrombin time when administered to patients receiving chronic warfarin treatment. **Endocrine Function** — HMG-CoA reductase inhibitors interfere with cholesterol synthesis and theoretically might blunt adrenal and/or gonadal steroid production. Clinical studies have shown that atorvastatin does not reduce basal plasma cortisol concentration or impair adrenal reserve. The effects of HMG-CoA reductase inhibitors on male fertility have not been studied in adequate numbers of patients. The effects, if any, on the pituitary-gonadal axis in premenopausal women are unknown. Caution should be exercised if an HMG-CoA reductase inhibitor is administered concomitantly with drugs that may decrease the levels or activity of endogenous steroid hormones, such as ketoconazole, spiroclonolone, and cimetidine. **CNS Toxicity** — Brain hemorrhage was seen in a female dog treated for 3 months at 120 mg/kg/day. Brain hemorrhage and optic nerve vacuolation were seen in another female dog that was sacrificed in moribund condition after 11 weeks of escalating doses up to 280 mg/kg/day. The 120 mg/kg dose resulted in a systemic exposure approximately 16 times the human plasma area-under-the-curve (AUC, 0-24 hours) based on the maximum human dose of 80 mg/day. A single tonic convulsion was seen in each of 2 male dogs (one treated at 10 mg/kg/day and one at 120 mg/kg/day) in a 2-year study. No CNS lesions have been observed in mice after chronic treatment for up to 2 years at doses up to 400 mg/kg/day or in rats at doses up to 100 mg/kg/day. These doses were 6 to 11 times (mouse) and 8 to 15 times (rat) the human AUC (0-24) based on the maximum recommended human dose of 80 mg/day. CNS vascular lesions, characterized by perivascular hemorrhages, edema, and mononuclear cell infiltration of perivascular spaces, have been observed in dogs treated with other members of this class. A chemically similar drug in this class produced optic nerve degeneration (Wallenrod degeneration of retinogeniculate fibers) in clinically normal dogs in a dose-dependent fashion at a dose that produced plasma drug levels about 30 times higher than the mean drug level in humans taking the highest recommended dose. **Carcinogenesis, Mutagenesis, Impairment of Fertility** — In a 2-year carcinogenicity study in rats at dose levels of 10, 30, and 100 mg/kg/day, 7 rare tumors were found in muscle in high-dose females. In one, there was a rhabdomyosarcoma and, in another, there was a fibrosarcoma. This dose represents a plasma AUC (0-24) value of approximately 16 times the mean human plasma drug exposure after an 80 mg oral dose. A 2-year carcinogenicity study in mice given 100, 200, or 400 mg/kg/day resulted in a significant increase in liver adenomas in high-dose males and liver carcinomas in high-dose females. These findings occurred at plasma AUC (0-24) values of approximately 6 times the human plasma drug exposure after an 80 mg oral dose. *In vitro*, atorvastatin was not mutagenic or clastogenic in the following tests with and without metabolic activation: the Ames test with *Salmonella typhimurium* and *Escherichia coli*, the HGPRT forward mutation assay in Chinese hamster lung cells, and the chromosomal aberration assay in Chinese hamster lung cells. Atorvastatin was negative in the *in vivo* mouse micronucleus test. Studies in rats performed at doses up to 175 mg/kg (15 times the human exposure) produced no changes in fertility.

There was aplasia and aspermia in the epididymis of 2 of 10 rats treated with 100 mg/kg/day of atorvastatin for 8 months (16 times the human AUC at the 80 mg dose); testis weights were significantly lower at 30 and 100 mg/kg and epididymal weight was lower at 100 mg/kg. Male rats given 100 mg/kg/day for 11 weeks prior to mating had decreased sperm motility, sperm head concentration, and increased abnormal sperm. Atorvastatin caused no adverse effects on semen parameters, or reproductive organ histopathology in dogs given doses of 10, 40, or 120 mg/kg for two years. **Pregnancy** — **Pregnancy Category X: See CONTRAINDICATIONS.** Safety in pregnant women has not been established. Atorvastatin crosses the rat placenta and reaches a level in fetal liver equivalent to that of maternal plasma. Atorvastatin was not teratogenic in rats at doses up to 300 mg/kg/day or in rabbits at doses up to 100 mg/kg/day. These doses resulted in multiples of about 30 times (rat) or 20 times (rabbit) the human exposure based on surface area (mg/m²). In a study in rats given 20, 100, or 225 mg/kg/day, from gestation day 7 through to lactation day 21 (weaning), there was decreased pup survival at birth, neonate, weaning, and maturity in pups of mothers dosed with 225 mg/kg/day. Body weight was decreased on days 1 and 21 in pups of mothers dosed at 100 mg/kg/day; pup body weight was decreased at birth and at days 4, 21, and 91 at 225 mg/kg/day. Pup development was delayed (rotarod performance at 100 mg/kg/day and acoustic startle at 225 mg/kg/day; pinnae detachment and eye opening at 225 mg/kg/day). These doses correspond to 6 times (100 mg/kg) and 22 times (225 mg/kg) the human AUC at 80 mg/day. Rare reports of congenital anomalies have been received following intrauterine exposure to HMG-CoA reductase inhibitors. There has been one report of severe congenital bony deformity, tracheo-esophageal fistula, and anal atresia (VATER association) in a baby born to a woman who took lovastatin with dextroamphetamine sulfate during the first trimester of pregnancy. **LIPITOR should be administered to women of childbearing potential only when such patients are highly unlikely to conceive and have been informed of the potential hazards.** If the woman becomes pregnant while taking LIPITOR, it should be discontinued and the patient advised again as to the potential hazards to the fetus. **Nursing Mothers** — Nursing rat pups had plasma and liver drug levels of 50% and 40%, respectively, of that in their mother's milk. Because of the potential for adverse reactions in nursing infants, women taking LIPITOR should not breastfeed (see CONTRAINDICATIONS). **Pediatric Use** — Treatment experience in a pediatric population is limited to doses of LIPITOR up to 80 mg/day for 1 year in 8 patients with homozygous FH. No clinical or biochemical abnormalities were reported in these patients. None of these patients was below 9 years of age. **Geriatric Use** — Treatment experience in adults age ≥70 years with doses of LIPITOR up to 80 mg/day has been evaluated in 221 patients. The safety and efficacy of LIPITOR in this population were similar to those of patients <70 years of age.

ADVERSE REACTIONS: LIPITOR is generally well tolerated. Adverse reactions have usually been mild and transient. In controlled clinical studies of 2,502 patients, <2% of patients were discontinued due to adverse experiences attributable to atorvastatin. The most frequent adverse events thought to be related to atorvastatin were constipation, flatulence, dyspepsia, and abdominal pain. **Clinical Adverse Experiences** — Adverse experiences reported in >2% of patients in placebo-controlled clinical studies of atorvastatin, regardless of causality assessment, are shown in the following table.

Adverse Events in Placebo-Controlled Studies (% of Patients)					
BODY SYSTEM	Placebo	Atorvastatin	Atorvastatin	Atorvastatin	Atorvastatin
Adverse Event		10 mg	20 mg	40 mg	80 mg
	N = 270	N = 863	N = 36	N = 79	N = 94
BODY AS A WHOLE					
Infection	10.3	10.3	2.8	10.1	7.4
Headache	7.0	5.4	16.7	2.5	6.4
Accidental Injury	3.7	4.2	0.0	1.3	3.2
Flu Syndrome	1.9	2.2	0.0	2.5	3.2
Abdominal Pain	0.7	2.8	0.0	3.8	2.1
Back Pain	3.0	2.8	0.0	3.8	1.1
Allergic Reaction	2.8	0.9	2.8	1.3	0.0
Asthenia	1.9	2.2	0.0	3.8	0.0
DIGESTIVE SYSTEM					
Constipation	1.8	2.1	0.0	2.5	1.1
Diarrhea	1.5	2.7	0.0	3.8	5.3
Dyspepsia	4.1	2.3	2.8	1.3	2.1
Flatulence	3.3	2.1	2.8	1.3	1.1
RESPIRATORY SYSTEM					
Sinusitis	2.6	2.8	0.0	2.5	6.4
Pharyngitis	1.5	2.5	0.0	1.3	2.1
SKIN AND APPENDAGES					
Rash	0.7	3.9	2.8	3.8	1.1
MUSCULOSKELETAL SYSTEM					
Arthralgia	1.5	2.0	0.0	5.1	0.0
Myalgia	1.1	3.2	5.6	1.3	0.0

The following adverse events were reported, regardless of causality assessment in patients treated with atorvastatin in clinical trials. The events in italics occurred in >2% of patients and the events in plain type occurred in <2% of patients.

Body as a Whole: Chest pain, face edema, fever, neck rigidity, malaise, photosensitivity reaction, generalized edema. **Digestive System:** Nausea, gastroenteritis, liver function abnormal, colitis, vomiting, gastritis, dry mouth, *hemorrhage*, esophagitis, eructation, glossitis, mouth ulceration, *anorexia*, increased appetite, *abnormal* biliary pain, chelitis, duodenal ulcer, dysphagia, enteritis, melena, gum hemorrhage, stomach ulcer, tenesmus, ulcerative stomatitis, hepatitis, pancreatitis, cholestatic jaundice. **Respiratory System:** Bronchitis, rhinitis, pneumonia, dyspnea, asthma, epistaxis. **Nervous System:** Insomnia, dizziness, paresthesia, somnolence, amnesia, abnormal dreams, libido decreased, emotional lability, incoordination, peripheral neuropathy, vertigo, facial paralysis, hyperkinesia, depression, hyposthesia, hyperaesthesia. **Musculoskeletal System:** Arthritis, leg cramps, bursitis, tenosynovitis, myofascitis, tendinous contracture, myositis. **Skin and Appendages:** Pruritus, contact dermatitis, alopecia, dry skin, sweating, acne, urticaria, eczema, seborrhea, skin ulcer. **Urogenital System:** Urinary tract infection, urinary frequency, cystitis, hematuria, impotence, dysuria, kidney calculus, nocturia, epididymitis, fibrocystic breast, vaginal hemorrhage, albuminuria, breast enlargement, metrorrhagia, nephritis, urinary incontinence, urinary retention, urinary urgency, abnormal ejaculation, uterine hemorrhage. **Special Senses:** Amblyopia, limbitis, dry eyes, refractive disorder, eye hemorrhage, deafness, glaucoma, parosmia, taste loss, taste perversion. **Cardiovascular System:** Palpitation, vasodilation, syncope, migraine, postural hypotension, phlebitis, arrhythmia, angina pectoris, hypertension. **Metabolic and Nutritional Disorders:** Peripheral edema, hyperglycemia, creatine phosphokinase increased, gout, weight gain, hypoglycemia. **Hemic and Lymphatic System:** Echinomiasis, anemia, lymphadenopathy, thrombocytopenia, petechia. **Postintroduction Reports** — Adverse events associated with LIPITOR therapy reported since market introduction, that are not listed above, regardless of causality assessment, include the following: anaphylaxis, angioneurotic edema, bulkus rashes (including erythema multiforme), Stevens-Johnson syndrome, and toxic epidermal necrolysis, and rhabdomyolysis.

OVERDOSAGE: There is no specific treatment for atorvastatin overdosage. In the event of overdosage, the patient should be treated symptomatically, and supportive measures instituted as required. Due to extensive drug binding to plasma proteins, hemodialysis is not expected to significantly enhance atorvastatin clearance.

This summary provides important information about Lipitor. For more information, please ask your doctor, pharmacist or healthcare professional to provide the professional labeling and then discuss it with them.

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RESTORATION

Saving Sculpture in a Big Way

Heritage Preservation helps public art get a face-lift

He was 71 years old, had had his head struck by lightning a few times (right), and was slowly eroding on his 400-foot-high perch. That's why "The Sower," the 19,000-pound, 19-foot-tall bronze statue atop the Nebraska State Capitol (above, with architect Mike Rindone) qualified for

a conservation grant from the Save Outdoor Sculpture! (SOS!) program of the Heritage Preservation Society. Some 7,000 SOS! volunteers, including Girl Scouts, garden clubs, and Civil War reenactors, identified more than 32,000 outdoor sculptures all over the U.S., half of which were in need of refurbishment.



PHOTO BY JOEL ROBERTSON

"Many communities aren't sure who is responsible for upkeep of sculpture, so it's never done," says SOS! director Susan Nichols. "We hope we can help this art receive proper maintenance and have its life extended."

ANIMAL BEHAVIOR

Caller ID: The Birds

If you hear a cell phone ring outdoors, you may be startled to discover that the "phone" has feathers.

Master mimics, starlings were taught by the Romans to imitate human speech. Today their repertoire includes not only other birdcalls but also sirens, chain saws, and horses whinnying.



ART BY JOHN BROWN

Now they've added the warbling of cell phones. Starlings as far apart as Denmark and Australia are learning the new trick.

Males re-create human sounds "especially at breeding time to attract a mate and hold territories," says Andrew South of Britain's Royal Society for the Protection of Birds. Starlings may not be alone. As cell phones proliferate, mockingbirds, mynahs, and other mimics are likely to get into the act.



It's not often a two-hour ride lasts a lifetime.

Talk to most motorcyclists, and they'll tell you how one ride can get inside you, and stay with you forever. This same phenomenon happens on another level at every *Ride for Kids* event. Each ride works to improve the lives of kids who have been diagnosed with childhood brain tumors by raising funds for medical research.

Our progress is encouraging. In 1984, a child diagnosed with a brain tumor was expected to live only five more months. Today, medical advancements allow that child to live another three years.

With Honda's support, more than 10,000 motorcyclists devote their time, money and energy to the *Ride for Kids* program every year. Yet everyone's goal of finding the cause of childhood brain tumors, and discovering a cure, remains many rides away. Which is why Honda continues to nurture this program. Because, while the ride lasts only a day, it leaves us with hope that lasts a lifetime.

HONDA

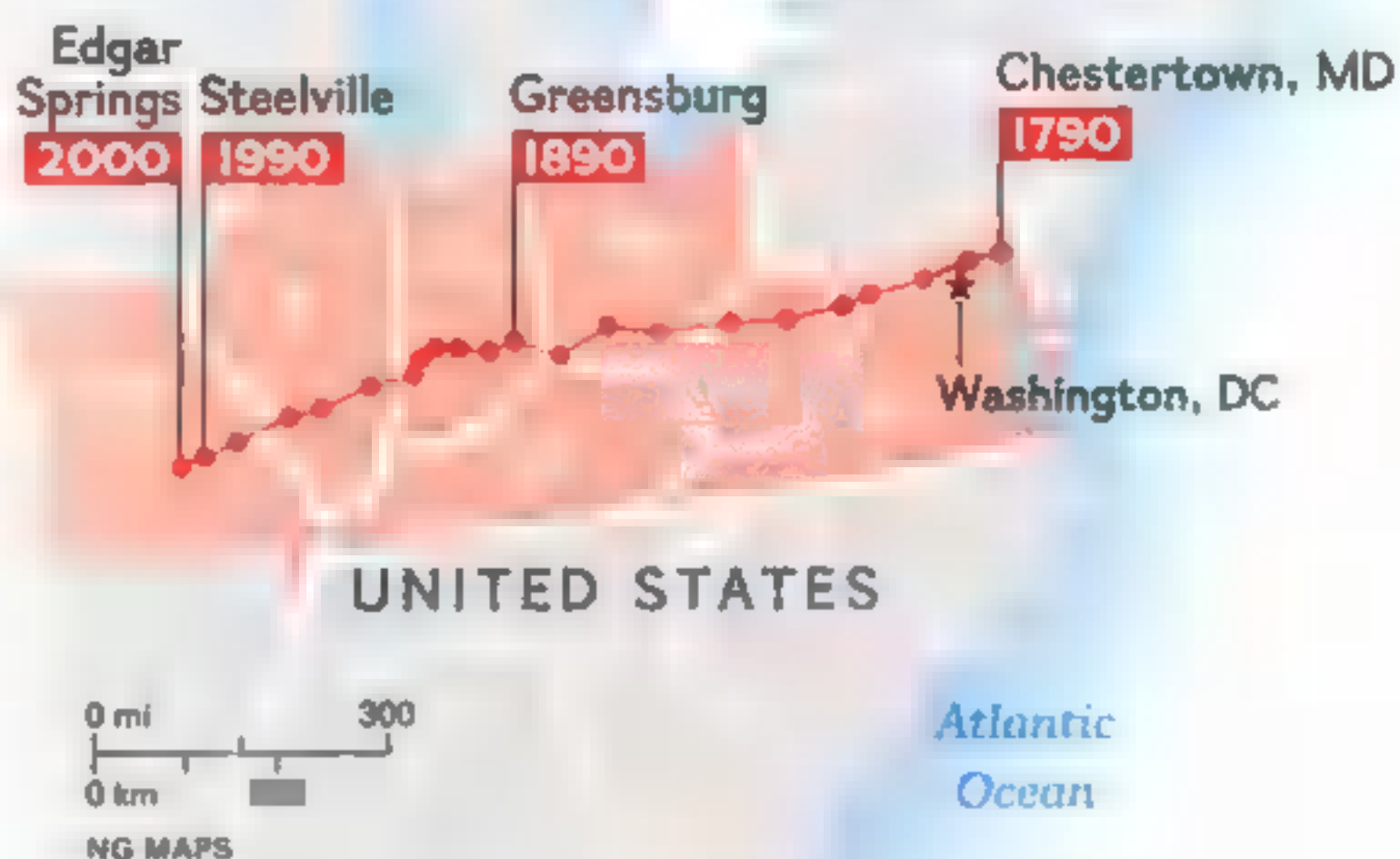
The power of dreams.

POPULATION

A Nation on the Move

Tiny Missouri town now claims the title of United States' population center

Just 190 people lived in Edgar Springs, Missouri, in the year 2000. But the little town is a big deal for the U.S. Census Bureau, which declared the town the country's newest population center, replacing Steelville. The formula for determining this is complex, figuring in residency distribution and latitude and longitude to find a hypothetical "human center of gravity" for the nation. The Census Bureau has calculated this center back to 1790, and the location has always moved west. "The value of the population center is similar to figures like average income or average age," says the bureau's Frederick Broome. "It's a broad indicator of the direction our population is moving."



NATURE WORLD

Hanging On for Dear Life

Predators rarely pry superbeetle loose

"Our little athlete's foot," Cornell University researcher Thomas Eisner calls the blue tortoise beetle. The mighty insect lives on palmetto fronds, where ants try to overturn it. When the beetle clamps its "feet," with 60,000 adhesive bristles, onto a frond, the ants can't budge it. How strong is it? This one withstands a two-gram pull—150 times its weight.



MARCO GUERRA, SMITHSONIAN TROPICAL RESEARCH INST

NGS RESEARCH GRANT

Clues to Frog Colors

Bright hues of poison-dart frogs advertise the frogs' toxicity. But in Panama one species, *Dendrobates pumilio*, sports an astonishing variety of colors that don't correlate with poison levels. Nor is there enough genetic divergence to explain the different tints, says East Carolina University biologist Kyle Summers. He also rules out habitat; two other poison-dart species share the same range yet do not vary in color. *D. pumilio* differs in that females do the parenting, which often goes with strong preferences in male ornamentation. Summers' theory: Females may pass along preferred colors through sexual selection.

THOMAS EISNER



Photographed by Zsolt Kabócsa

WILDLIFE AS CANON SEES IT

From its songpost in the sedge fen mires of Hungary's Hortobágyi National Park, a male aquatic warbler sings loudly to attract females. The males' distinctive songs ring out at dawn and dusk throughout the breeding season. Females, meanwhile, incubate and care for their two annual broods alone, relying on a nearby abundance of large insects and spiders to feed their young. These warblers generally stay out of sight, skulking quietly down around low vegetation. Breeding habitat has dwindled for decades throughout Europe. With the loss of these

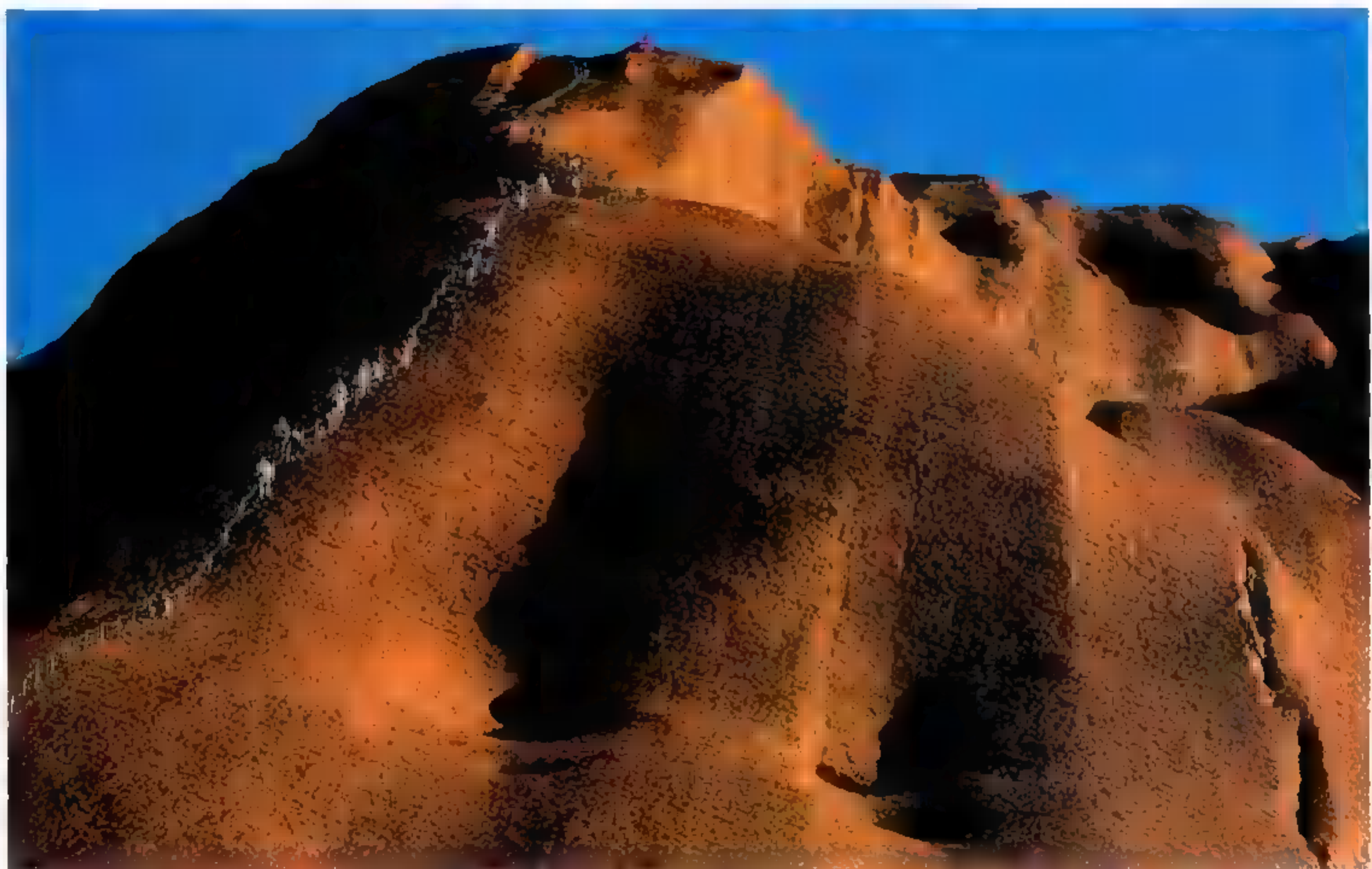
vital wetland areas, the aquatic warbler population has declined dramatically.

As a global corporation committed to social and environmental concerns, we join in worldwide efforts to promote greater awareness of endangered species for the benefit of future generations.



Aquatic Warbler (*Acrocephalus paludicola*)
Size: Length, 12-13 cm
Weight: Approx. 12 g
Habitat: Breeds in lowland marsh habitats in Europe (and western Siberia, where its range is little known); migrates to tropical West Africa in winter
Surviving number: European population estimated at 13,500-21,000 singing males





PAUL CHESLEY

INDIGENOUS PEOPLES

No Walk in the Park

Australian Aborigines object to climbers on Uluru

Visitors have been drawn to climb Ayers Rock, now known by its Aboriginal name of Uluru, since the first road reached it in 1948. But the massive sandstone monolith looming over the flat Australian desert is a sacred site to the indigenous Anangu population.

Scaling the 1,100-foot rock is, to them, a kind of blasphemy.

Uluru is located in central Australia, in Uluru-Kata Tjuta National Park. Some 500,000 people travel to its remote location every year, and perhaps half choose to climb to the rock's summit (above) even though

signs in the park request that they not. After the death of an Anangu elder in May 2001, Uluru's climbing path was closed for ten days during a mourning period. Fearing that tribal leaders might seek a permanent ban, the region's tourist industry protested. The park has stepped up educational efforts to persuade visitors not to ascend.

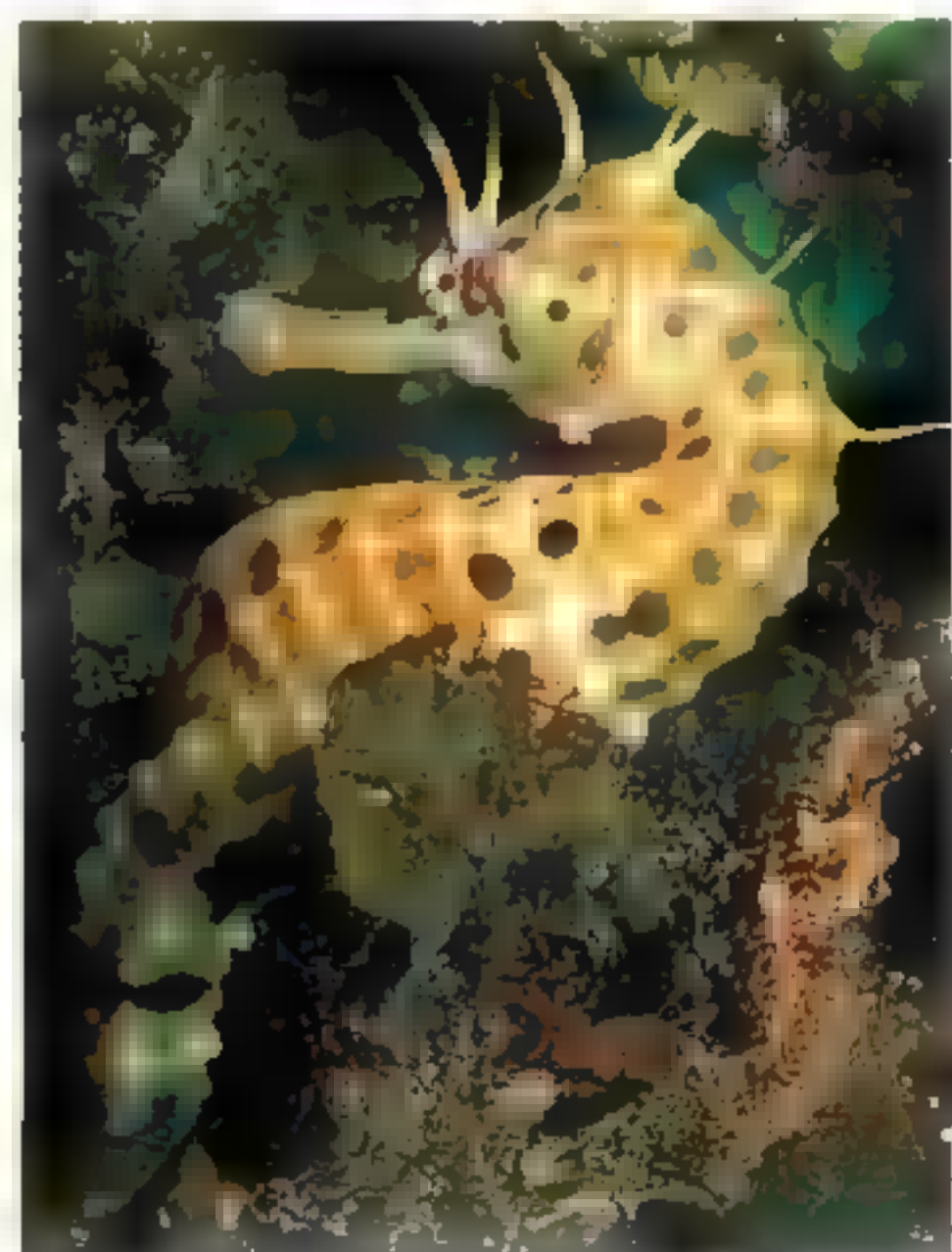
Respect for Aboriginal beliefs is not the only reason to stay off Uluru. So far, 35 people have died while making the climb.

CONSERVATION

Keeping Tabs on the Seahorse Trade

More than 20 million seahorses are plucked from the world's oceans each year. Many are dried and sold as curios or for traditional medicines; live ones adorn aquariums. Progress in

managing the trade has been made, thanks to McGill University biologist Amanda Vincent (*GEOGRAPHIC*, October 1994) and her colleagues, who founded Project Seahorse. In 1998 Australia classed seahorses under its Wildlife Protection Act and requires export permits. Hong Kong, Taiwan, and the European Union also monitor the trade. In the Philippines, Project Seahorse is helping set up sanctuaries where no fishing is allowed.



GEORGE GRALL

> If ever there was a time to get all our energy from one source, this isn't it.



ENERGY: WHAT'S EMERGING The estimates are hair-raising. By 2020, the U.S. will need 45% more electricity. And it's the little things that are hurting us: Your home PC sucks up half as much energy as your refrigerator. Clearly, no one fuel can answer the demand. Hence, Exelon's diversified energy portfolio. We're generating nuclear energy more efficiently than ever. We're researching solar power. And soon, we'll be the East's largest marketer of wind energy. Because there's no solution to the energy problem. There are several. [More at exeloncorp.com](http://www.exeloncorp.com)

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Behind the SCENES

AT THE NATIONAL GEOGRAPHIC SOCIETY

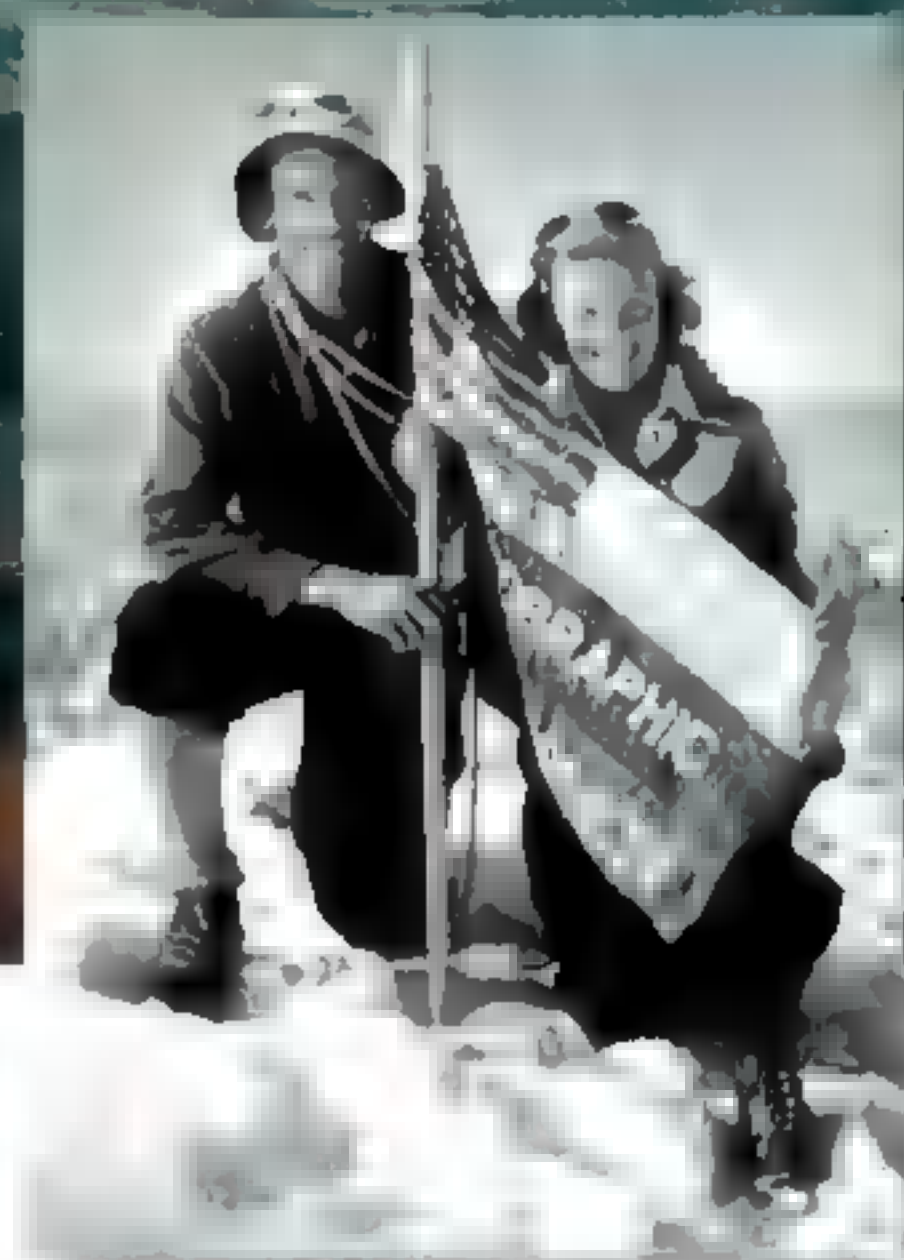
EXPLORERS-IN-RESIDENCE

Yet Another Pinnacle

Expeditions Council honors intrepid Washburns

In March 1930 Bradford Washburn, a 19-year-old mountaineer, gave a Society lecture to reveal a new trail he had forged up an Alpine peak. Last year Brad, now 91, and his wife, Barbara, were named honorary explorers-in-residence by our Expeditions Council.

Three months after marrying in 1940, Brad—founding director of Boston's Museum of Science—and Barbara climbed their first mountain together, the first ascent of Alaska's Mount Bertha (inset). In 1947 Barbara became the first woman to summit Mount McKinley, and in the



1970s the couple surveyed the heart of the Grand Canyon for the magazine (top). Brad's latest grant, in 2001, funded a study of snow depth on Mount Everest.

Close-up on History

An original proclamation of the Louisiana Purchase (right), signed by Thomas Jefferson and James Madison, is on display in Omaha's Joslyn Art Museum to mark the recent premiere of a National Geographic large-format film, *Lewis & Clark: Great Journey West*. Omaha businessman Walter Scott, Jr., acquired the document—in private hands since the 19th century—in 1995. Scott regards the 1803 Louisiana Purchase as “the



COLLECTION OF WALTER SCOTT, JR.; PHOTOGRAPH BY INA BLOCK; DAVID SIEGEL

most important single event in American history.” National Geographic Television & Film produced the 40-minute film

in association with the Suzanne and Walter Scott Foundation. It will appear soon in giant-screen theaters worldwide.

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Please see additional important information about Nasacort AQ on next page.



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ONCE DAILY Nasacort AQ[™] (triamcinolone acetonide) Nasal Spray

For intranasal use only.
Shake Well Before Using

BRIEF SUMMARY

CONTRAINDICATIONS

Hypersensitivity to any of the ingredients of this preparation contraindicates its use.

WARNINGS

The replacement of a systemic corticosteroid with a topical corticosteroid can be accompanied by signs of adrenal insufficiency and, in addition, some patients may experience symptoms of withdrawal; e.g., joint and/or muscular pain, lassitude and depression. Patients previously treated for prolonged periods with systemic corticosteroids and transferred to topical corticosteroids should be carefully monitored for acute adrenal insufficiency in response to stress. In those patients who have asthma or other clinical conditions requiring long-term systemic corticosteroid treatment, too rapid a decrease in systemic corticosteroids may cause an exacerbation of their symptoms.

Children who are on immunosuppressant drugs are more susceptible to infections than healthy children. Chickenpox and measles, for example, can have a more serious or even fatal course in children on immunosuppressant doses of corticosteroids. In such children, or in adults who have not had these diseases, particular care should be taken to avoid exposure. If exposed, therapy with varicella-zoster immune globulin (VZIG) or pooled intravenous immunoglobulin (IVIG), as appropriate, may be indicated. If chickenpox develops, treatment with antiviral agents may be considered.

PRECAUTIONS

General: In clinical studies with triamcinolone acetonide nasal spray, the development of localized infections of the nose and pharynx with *Candida albicans* has rarely occurred. When such an infection develops it may require treatment with appropriate local or systemic therapy and discontinuance of treatment with Nasacort AQ Nasal Spray.

Nasacort AQ Nasal Spray should be used with caution, if at all, in patients with active or quiescent tuberculous infection of the respiratory tract or in patients with untreated fungal, bacterial, or systemic viral infections or ocular herpes simplex.

Because of the inhibitory effect of corticosteroids, in patients who have experienced recent nasal septal ulcers, nasal surgery, or trauma, a corticosteroid should be used with caution until healing has occurred. As with other nasally inhaled corticosteroids, nasal septal perforations have been reported in rare instances.

When used at excessive doses, systemic corticosteroid effects such as hypercorticism and adrenal suppression may appear. If such changes occur, Nasacort AQ Nasal Spray should be discontinued slowly, consistent with accepted procedures for discontinuing oral steroid therapy.

Information for Patients: Patients being treated with Nasacort AQ Nasal Spray should receive the following information and instructions. Patients who are on immunosuppressant doses of corticosteroids should be warned to avoid exposure to chickenpox or measles and, if exposed, to obtain medical advice.

Patients should use Nasacort AQ Nasal Spray at regular intervals since its effectiveness depends on its regular use. (See **DOSE AND ADMINISTRATION**.)

An improvement in some patient symptoms may be seen within the first day of treatment, and generally, it takes one week of treatment to reach maximum benefit. Initial assessment for response should be made during this time frame and periodically until the patient's symptoms are stabilized.

The patient should take the medication as directed and should not exceed the prescribed dosage. The patient should contact the physician if symptoms do not improve after three weeks, or if the condition worsens. Patients who experience recurrent episodes of epistaxis (nose bleeds) or nasal septum discomfort while taking this medication should contact their physician. For the proper use of this unit and to attain maximum improvement, the patient should read and follow the accompanying patient instructions carefully.

It is important to shake the bottle well before each use. Also, the bottle should be discarded after 120 actuations since the amount of triamcinolone acetonide delivered thereafter per actuation may be substantially less than 55 mcg of drug. Do not transfer any remaining suspension to another bottle.

Carcinogenesis, Mutagenesis, and Impairment of Fertility: In a two-year study in rats, triamcinolone acetonide caused no treatment-related carcinogenicity at oral doses up to 1.0 mcg/kg (approximately 1/30 and 1/50 of the maximum recommended daily intranasal dose in adults and children on a mcg/m² basis, respectively). In a two-year study in mice, triamcinolone acetonide caused no treatment-related carcinogenicity at oral doses up to 3.0 mcg/kg (approximately 1/12 and 1/30 of the maximum recommended daily intranasal dose in adults and children on a mcg/m² basis, respectively).

No mutagenicity studies with triamcinolone acetonide have been performed.

In male and female rats, triamcinolone acetonide caused no change in pregnancy rate at oral doses up to 15.0 mcg/kg (approximately 1/2 of the maximum recommended daily intranasal dose in adults on a mcg/m² basis). Triamcinolone acetonide caused increased fetal resorptions and stillbirths and decreases in pup weight and survival at doses of 5.0 mcg/kg and above (approximately 1/5 of the maximum recommended daily intranasal dose in adults on a mcg/m² basis). At 1.0 mcg/kg (approximately 1/30 of the maximum recommended daily intranasal dose in adults on a mcg/m² basis), it did not induce the above mentioned effects.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Triamcinolone acetonide was teratogenic in rats, rabbits, and monkeys. In rats, triamcinolone acetonide was teratogenic at inhalation doses of 20 mcg/kg and above (approximately 7/10 of the maximum recommended daily intranasal dose in adults on a mcg/m² basis). In rabbits, triamcinolone acetonide was teratogenic at inhalation doses of 20 mcg/kg and above

(approximately 2 times the maximum recommended daily intranasal dose in adults on a mcg/m² basis). In monkeys, triamcinolone acetonide was teratogenic at an inhalation dose of 500 mcg/kg (approximately 37 times the maximum recommended daily intranasal dose in adults on a mcg/m² basis). Dose-related teratogenic effects in rats and rabbits included cleft palate and/or internal hydrocephaly and axial skeletal defects, whereas the effects observed in the monkey were cranial malformations.

There were no adequate and well-controlled studies in pregnant women. Therefore, triamcinolone acetonide should be used in pregnancy only if the potential benefit justifies the potential risk to the fetus. Since their introduction, experience with oral corticosteroids in pharmacologic as opposed to physiologic doses suggests that rodents are more prone to teratogenic effects from corticosteroids than humans. In addition, because there is a natural increase in glucocorticoid production during pregnancy, pregnant women will require a lower exogenous corticosteroid dose and many will not need corticosteroid treatment during pregnancy.

Nonteratogenic Effects: Hypoadrenalism may occur in infants born of mothers receiving corticosteroids during pregnancy. Such infants should be carefully observed.

Nursing Mothers: It is not known whether triamcinolone acetonide is excreted in human milk. Because other corticosteroids are excreted in human milk, caution should be exercised when Nasacort AQ Nasal Spray is administered to nursing women.

Use: Safety and effectiveness in pediatric patients below the age of 6 years have not been established.

Corticosteroids have been shown to cause growth suppression in children and teenagers, particularly with higher doses over extended periods. If a child or teenager on any corticosteroid appears to have growth suppression, the possibility that they are particularly sensitive to this effect of corticosteroids should be considered.

ADVERSE REACTIONS

In placebo-controlled, double-blind, and open-label clinical studies, 1483 adults and children 12 years and older received treatment with triamcinolone acetonide aqueous nasal spray. These patients were treated for an average duration of 51 days. In the controlled trials (2-5 weeks duration) from which the following adverse reaction data are derived, 1394 patients were treated with Nasacort AQ Nasal Spray for an average of 19 days. In a long-term, open-label study, 172 patients received treatment for an average duration of 286 days.

Adverse events occurring at an incidence of 2% or greater and more common among Nasacort AQ-treated patients than placebo-treated patients in controlled adult clinical trials were:

Adverse Events	Patients treated with 220 mcg triamcinolone acetonide (n=857) %	Vehicle Placebo (n=662) %
Pharyngitis	5.1	3.6
Epistaxis	2.7	0.8
Increase in cough	2.1	1.5

A total of 602 children 6 to 12 years of age were studied in 3 double-blind, placebo-controlled clinical trials. Of these, 172 received 110 mcg/day and 207 received 220 mcg/day of Nasacort AQ Nasal Spray for two, six, or twelve weeks. The longest average durations of treatment for patients receiving 110 mcg/day and 220 mcg/day were 76 days and 80 days, respectively. Only 1% of those patients treated with Nasacort AQ were discontinued due to adverse experiences. No patient receiving 110 mcg/day discontinued due to a serious adverse event and one patient receiving 220 mcg/day discontinued due to a serious event that was considered not drug related. Overall, these studies found the adverse experience profile for Nasacort AQ to be similar to placebo. A similar adverse event profile was observed in pediatric patients 6-12 years of age as compared to older children and adults with the exception of epistaxis which occurred in less than 2% of the pediatric patients studied.

Adverse events occurring at an incidence of 2% or greater and more common among adult patients treated with placebo than Nasacort AQ were: headache, and rhinitis. In children aged 6 to 12 years these events included: asthma, epistaxis, headache, infection, otitis media, sinusitis, and vomiting.

In clinical trials, nasal septum perforation was reported in one adult patient although relationship to Nasacort AQ Nasal Spray has not been established.

In the event of accidental overdose, an increased potential for these adverse experiences may be expected, but acute systemic adverse experiences are unlikely. (See **OVERDOSAGE**.)

OVERDOSAGE

Like any other nasally administered corticosteroid, acute overdosing is unlikely in view of the total amount of active ingredient present. In the event that the entire contents of the bottle were administered all at once, via either oral or nasal application, clinically significant systemic adverse events would most likely not result. The patient may experience some gastrointestinal upset.

Caution: Federal law prohibits dispensing without prescription.

Please see product circular for full prescribing information.

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KENNETH WRIGHT

How Did Machu Picchu Work?

Studying the infrastructure of an Inca site

When Ruth Wright, a Colorado lawyer, first visited Machu Picchu with her daughters in 1974, she was, like most visitors, “blown away” by the legendary Inca site. But when she returned home, she was puzzled: How did the Inca get a water supply so high up on an Andean mountain?

After two decades spent seeking a permit that would allow them to investigate, Wright and her husband, Kenneth, a civil engineer (above), finally began to study the site’s infrastructure in 1994. They examined the layout of the buildings and how the fountains were designed. They also analyzed the types of roofs used and what crops the residents grew on the terraces. They ended up with the most detailed map ever made of Machu Picchu, where 16 fountains once

flowed, and gave invaluable assistance to our own cartographers in producing the supplement map in this issue.

“Basically, we’ve studied city planning there,” Ruth says. Ken adds: “We’ve studied hydrology, engineering, how it endured for 500 years without turning into a pile of rubble. The Inca created a functioning community without a written language, iron and steel, or the wheel.” The Wrights surveyed the site again and again, turning up information in some cases only after hacking away forest vegetation that had buried foundations, walls, and a trail.

So how did the Inca get their water? They built a canal to channel water from the natural spring on the mountain, the result of fractures in the underlying granite along the Machu Picchu fault.



A special-edition poster of Robert Clark’s photograph, featured on our January 2002 cover, is available for \$39.95 plus \$6.95 for postage and handling (\$9.95 for international orders). Part of the proceeds will benefit Vital Ground, an organization working to protect grizzly bear and wolf habitat (www.vitalground.org). Please add the appropriate sales tax for orders sent to CA, DC, FL, KY, MD, MI, PA, and Canada. We will produce only ■ many 24-by-30-inch prints as we receive orders for by May 31, 2002. Each will be hand-numbered and embossed with the Society seal. Shipping is scheduled for July 2002. To order, call toll free: 1-888-647-7301 (outside the U.S. and Canada call 1-515-362-3353).

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Cold Turkey?

A meat thermometer can measure how thoroughly this poultry has been cooked, but what else should you know about the bird your family consumes every day? Photographer Jim Richardson narrates a Sights & Sounds piece on food safety and the genetic modification of food. Eat carefully at nationalgeographic.com/sect/0205.

JIM RICHARDSON

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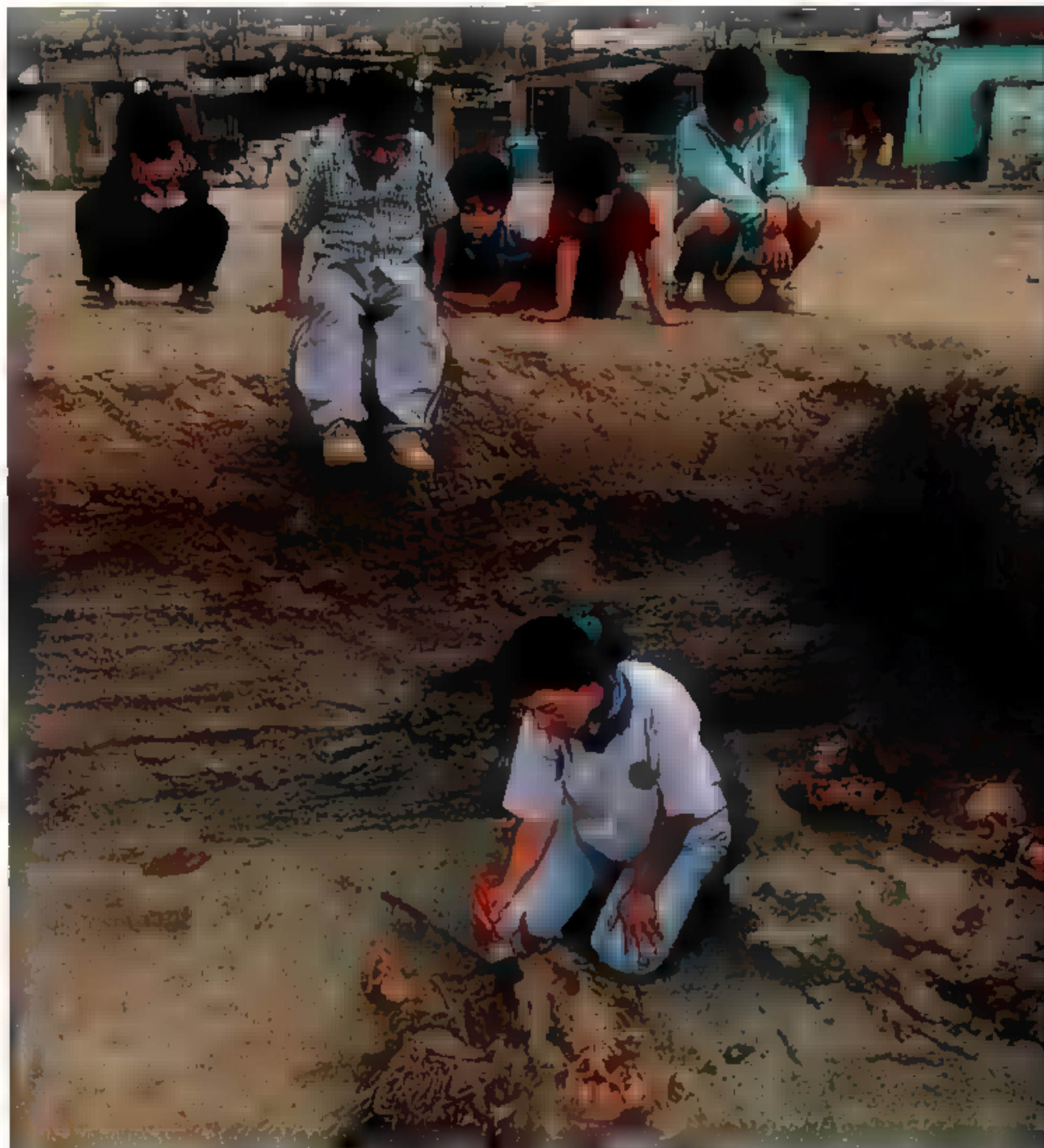
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Inca Rising

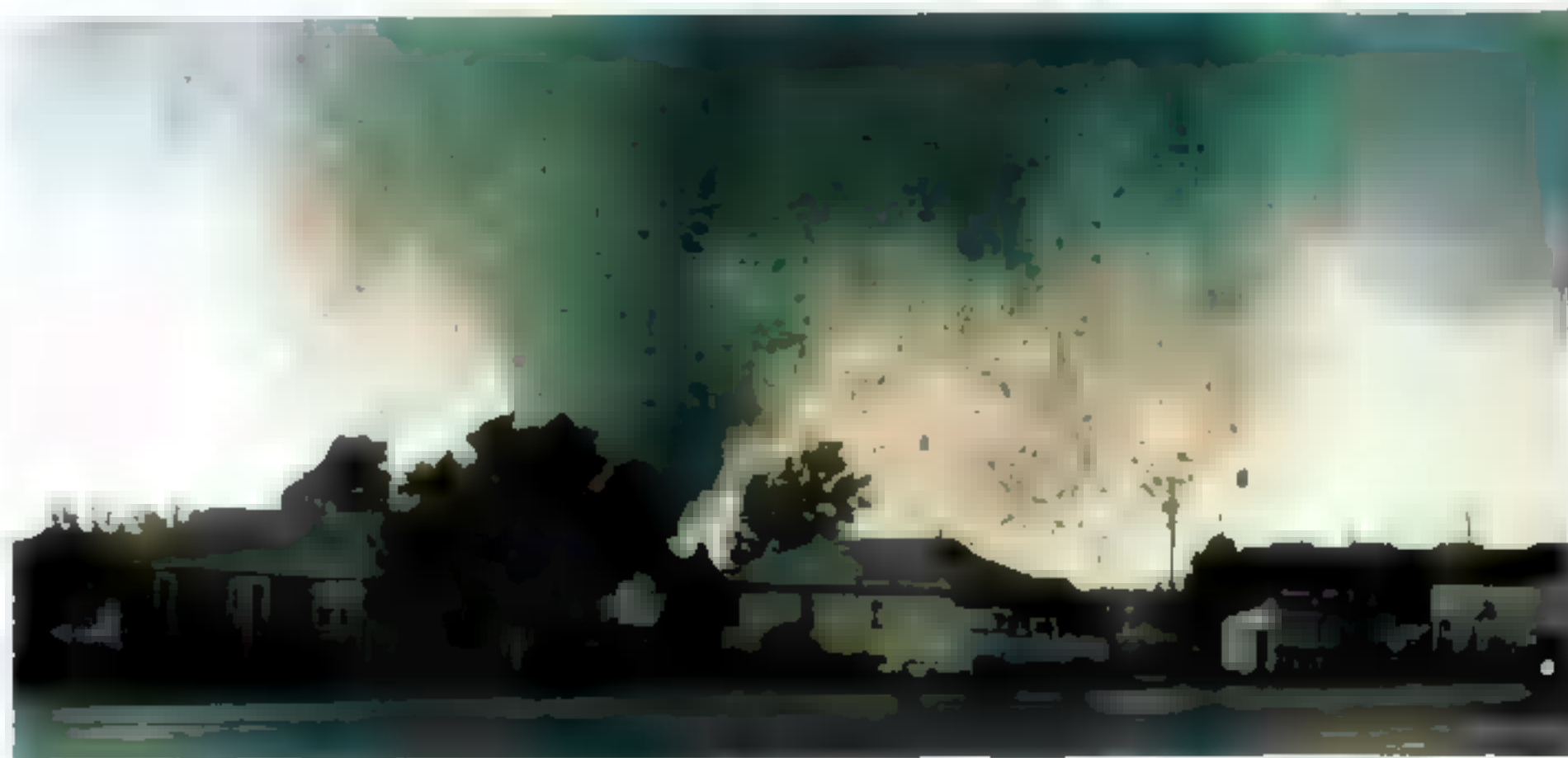
Inca rule extended along the western edge of South America through the early 16th century. Now almost 500 years after the empire fell to Spanish conquistadors, dramatic traces of the once powerful civilization come to light.

And Mummies: Secrets of a Lost World follows the efforts of two NGC-sponsored expeditions to unlock the secrets of ancient sites. On a ridge in the Andes, archaeologists study a newly discovered sacred center. And in a small town outside Peru's capital of Lima, scientists race to unearth mummies (left) before development erases the past.

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Into Rough Water

Competing in the world's most dangerous yachting race, six sailors struggle to survive a violent storm in *Catastrophe at Sea*.



TONY McDONOUGH/LEFT; MOLLER; GETTY IMAGES

NATIONAL GEOGRAPHIC EXPLORER, MSNBC

Chasing Down the Wind

Getting close enough to the funnel clouds to collect data—without getting killed—is the goal for a team of daredevil researchers featured in *Into the Tornado*.

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Ask Us

THE ANSWER PLACE

Our Research Correspondence staff responds to questions from curious readers.

Q Why are some ocean waves luminescent?

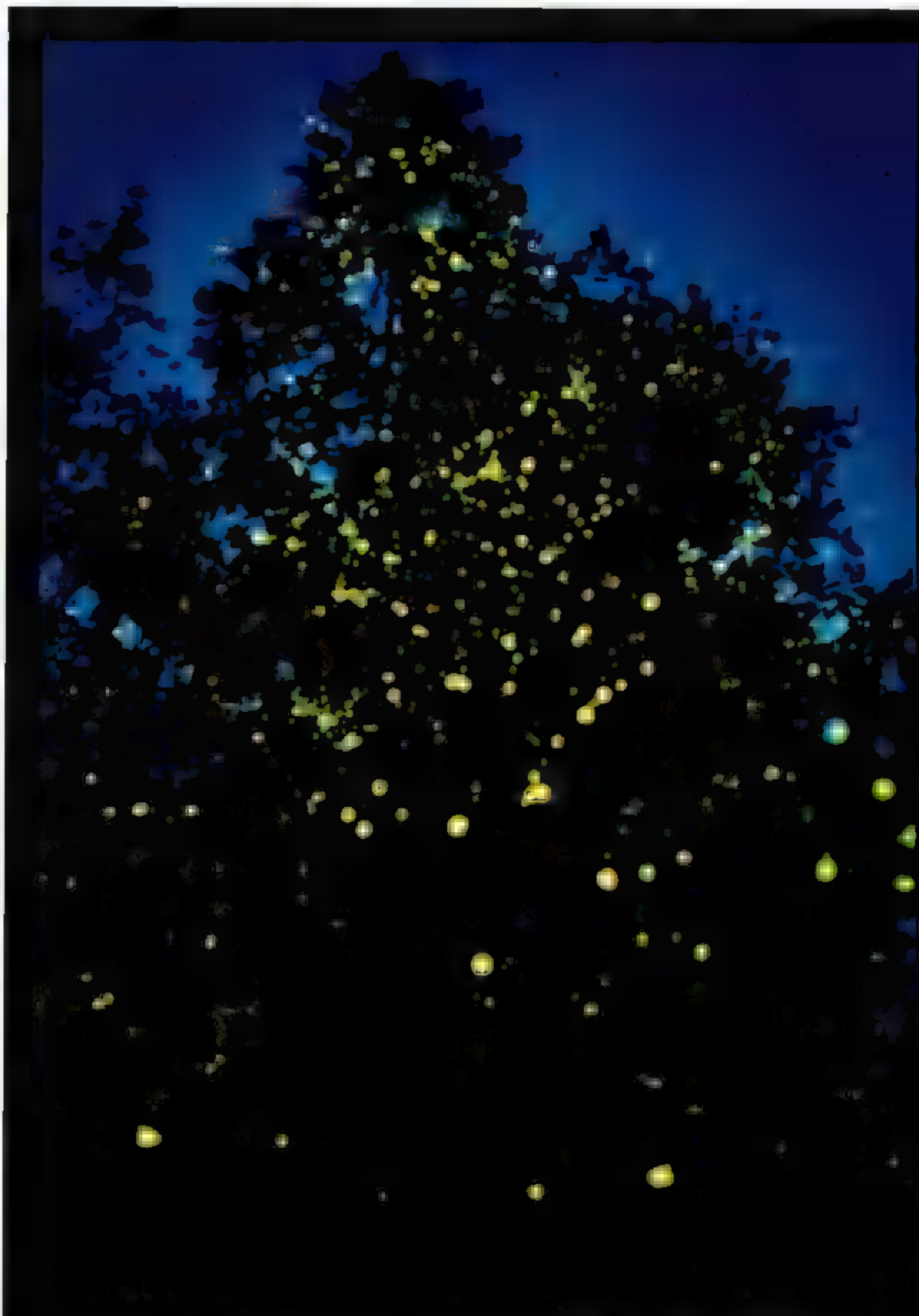
A The sea is full of bioluminescent single-cell life-forms known as dinoflagellates. If disturbed, they give off chemically produced light. This light creates auras around fish that feed on them, betraying the fish to predators. A ship can stir up so many dinoflagellates that its wake can be seen for miles.

Q How did North Carolina get the nickname Tar Heel State?

A There are various legends, all based on one of the state's earliest products, pine tar. One story is that during a Civil War battle some Confederate troops retreated, leaving a North Carolina regiment behind. The North Carolinians suggested putting some of their state's tar on the heels of other soldiers to make them stick around in a fight. Gen. Robert E. Lee is said to have commented, "God bless the Tar Heel boys"—and the nickname stuck.

MORE INFORMATION

Send questions to Ask Us, National Geographic Magazine, PO Box 98199, Washington, DC 20090-8199 or via the Internet to askus@nationalgeographic.com. Include name, address, and daytime phone number.



(VAN POLUNIN, BRUCE COLEMAN, PICTUREQUEST)

TELL US

What is a Christmas tree doing in a Malaysian forest? Hint: It's shy during the day and radiant at night.

Think you know the answer? Go online to nationalgeographic.com/ngm/tellus/0205 and test yourself, or read it here in next month's Issue.

April answer The shortnose batfish is charming to its prey. The bulb structure between its eyes lures down and lures smaller fish within striking distance.

Food

As this global industry expands and evolves, scientists and consumers are raising more and more questions. How can we keep our food supply safe? And what might be the benefits and risks of ever accelerating advances in genetic engineering?

By Jennifer Ackerman

Photographs by Jim Richardson

GROWING INDUSTRY

The time-honored act of planting by hand, though still practiced by millions in India and other developing nations, seems increasingly archaic in the face of big-business agriculture







HOT TOH

Cooked Right?

HOPING FOR THE BEST
Behind each order for a sausage-and-pepper sandwich at the New York State Fair in Syracuse lies a customer's trust that the food will not make him sick. State and county inspectors monitor meat temperatures and sanitary conditions. Yet guaranteeing the safety of meats and produce has become an increasingly complex and uncertain proposition as Americans more than ever depend on food produced and prepared outside the home.





Same Fish?

WHAT HAPPENS WHEN IT'S CHANGED?

Two salmon species are being raised in the same environment, and the results are surprising. One is a modified strain that grows at a faster pace, while the other grows more slowly in nature. As the world moves to bring fast-growing salmon to market, biologist Bob Devlin assesses the risk the fish would pose to natural populations if they escaped into the wild. "I've been working with these species for ten years, and I don't know."



How Safe?

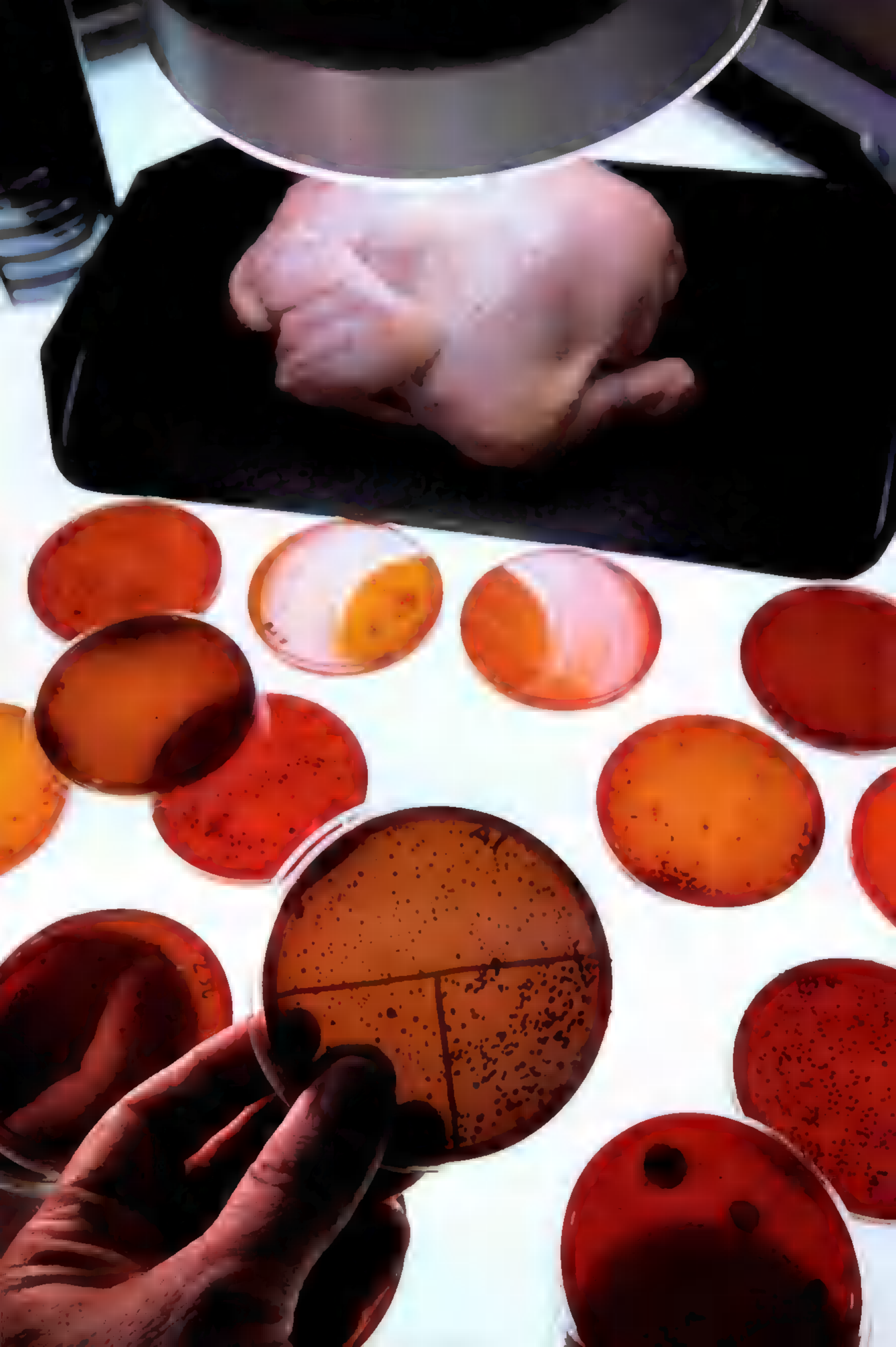
The United States boasts the safest food in the world. Maybe so, maybe not. Each year one in four citizens suffers from a foodborne illness, and some 5,000 people die from something they ate. From field to kitchen, risks crop up everywhere.

The chief topics of discussion one midsummer afternoon in a conference room at the Centers for Disease Control and Prevention (CDC) are ground beef, eggs, salad, almonds, and cilantro. This is no conversation about the lunch menu but a review of outbreaks nationwide of disease caused by food. At the table are 26 epidemiologists—medical detectives charged with investigating the mysterious links between contaminated food and the illness it causes.

The stories are not those I expect to hear, of people getting sick from drinking unpasteurized milk or eating deviled eggs left too long in the hot sun at a picnic, but tales of people sickened by contaminated parsley and scallions, cantaloupes, leaf lettuce, sprouts, orange juice, and almonds; refrigerated potato salad, eggs, chicken, salami, and beans; hot dogs, hamburgers, deli meats. The food culprits were served in kitchens, restaurants, and nursing homes, on cruise ships and farms, at churches and temples, family reunions, county fairs, casinos, day-care centers.

FRESH CONCERNS

Mushrooms from Canada, peppers from the Netherlands, squash from California. A global harvest supplies Seattle's Pike Place Market. With raw produce responsible for an increasing percentage of foodborne disease cases, experts implore consumers to wash all produce, no matter how good it looks.



They were distributed among many towns, in many states.

According to the CDC, each year in the United States 76 million people suffer from foodborne disease; 325,000 of them are hospitalized and 5,000 die. In the developing world contaminated food and water kill almost two million children a year. The epidemiologists in this room are keenly aware that behind the numbing, cold-potato statistics are real people, particularly the very young and the very old, who have suffered debilitating, even lethal, disease from what most of us consider one of life's less risky activities: eating.

On the face of it, it seems that "risk" should not be in the same sentence with "food"—that essential and wholesome component of life, so mixed and mingled with comfort, security, even love. But often it is. In recent years we've heard about the dangerous adulterants contaminating our food: pesticides on our grapes, carcinogens on our strawberries, chemicals on our apples, poisonous metals in our fish. We've heard dire warnings of the long-term effects of taking in too much fat or salt or cholesterol. In fact, in the past 30 years or so, there have been so many findings about the possible ill effects of our meals—some of them refuted shortly after being announced—that many of us have become inured to the red flags raised over food dangers.

I consider myself knowledgeable about safe eating. I thought I knew how to buy safe foods; how to clean, cook, and eat them properly; which dishes to order in restaurants and which to avoid. But the stories I have heard from food safety experts and the tales swapped among the epidemiologists at the CDC have

swept away my assumptions. I'm starting to rethink the way I shop, cook, eat, feed my children, even the way I define food and see its place in the world.

AMONG THE AGENCIES that oversee the safety of the U.S. food supply is the Department of Agriculture (USDA), charged with regulating meat and poultry and foods that contain them. It also regulates pasteurized egg products. The Food and Drug Administration (FDA) addresses the safety of all other foods, including fresh produce, canned and imported foods, milk, shell eggs, seafoods, and any processed foods that do not contain meat and poultry. These agencies post periodic alerts about hazards in food—chemical contaminants, food additives, unlabeled allergenic ingredients. These are matters of serious concern. But most government officials and health experts agree: The



Put Petri Dishes on Chickens

Petri dishes contain colonies of *Campylobacter*, ■ disease-causing bacterium found on retail chickens tested at the University of Arkansas. Proper cooking temperatures kill *Campylobacter*, yet those who prepare or eat the food can still get sick if raw poultry juice touches other food. To combat *Salmonella*, Arkansas graduate student Lisa Bielke sprays "healthful" bacteria onto chicks in an experiment to determine if those bacteria can out-compete harmful microbes in their intestinal tracts.

PRODUCTION

Raising tens of thousands of broiler chickens in a confined space exposes them to various threats of contamination. Infected fecal material in litter can contaminate the flock with *Campylobacter* and *Salmonella*. Rodents and wild birds can also introduce harmful bacteria.

Fruits and vegetables in the field can be tainted by runoff from uncomposted manure.

PROCESSING

At the slaughterhouse chickens whose intestines are accidentally pierced by gutting hooks are removed from the assembly line and processed elsewhere. But contaminated mechanical "fingers" used for plucking can press bacteria into skin. Pathogens may also spread in the chilled water baths in which carcasses are plunged.

Fruits and vegetables can be contaminated by polluted rinse water.

DISTRIBUTION

Usually packaged at the processing plant, chicken may spread contamination if juices leak in shipment. If chicken is improperly refrigerated at the store, bacteria will multiply. Studies show that a majority of store chickens contain pathogens.

Transporting fruits and vegetables in unsanitary trucks and storing them at improper temperatures also leads to contamination.



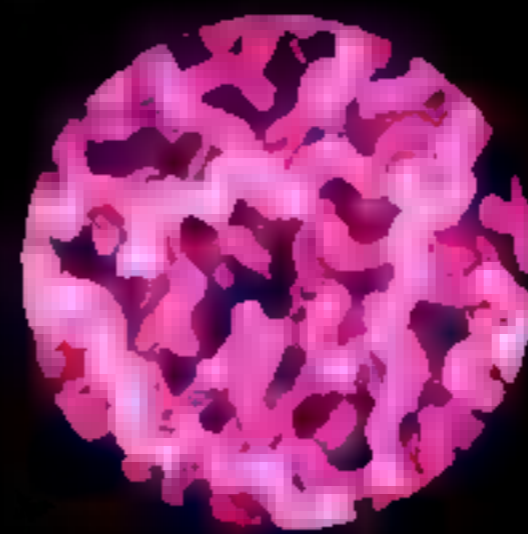
PREPARATION

Cooking chicken until juices run clear and the insides reach 180°F will kill harmful microbes. Problems can arise from cross-contamination. Raw juice on a cutting board or countertop can transfer pathogens to food prepared afterward. All surfaces and utensils that contact the juice should be washed in hot, soapy water. Raw fruits and vegetables should be thoroughly washed.

Farm to Fork

USUALLY WE CAN'T SEE, SMELL, OR TASTE THE BACTERIA THAT MAKE US SICK.

Whether people are getting sicker from food today than they were 50 years ago is a matter of debate. What is different are the pathogens. Advances in processing and sanitation in the developed world have diminished foodborne threats such as cholera and botulism, but new hazards come with imported foods, changes in the scale of food production, and the decline in home cooking.

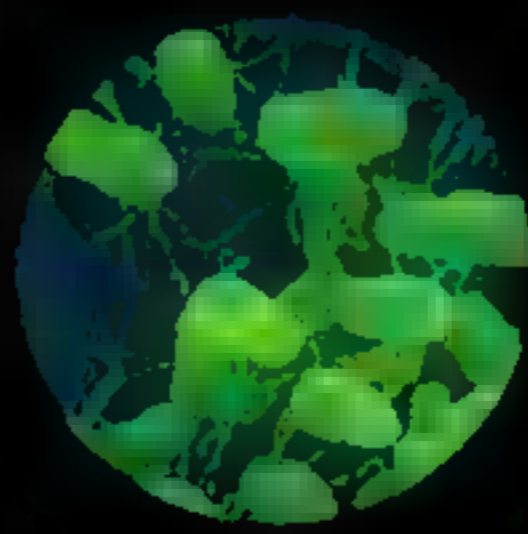
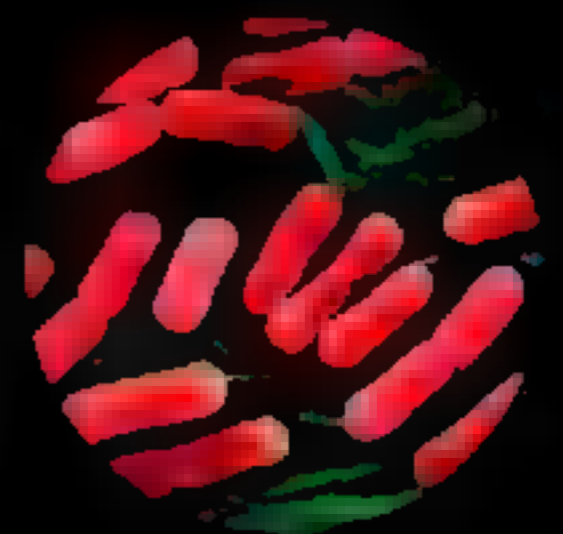


Campylobacter jejuni

Common in poultry flocks, this bacterium has become the leading cause of bacterial foodborne disease in the U.S. Exposure to a small number of microbes can cause illness. Most infected persons recover within a week.

Escherichia coli O157:H7

First identified as a cause of disease in 1982, this bacterium produces a powerful toxin that causes bloody diarrhea and occasionally kidney failure. Infections generally result from eating undercooked ground beef.



Salmonella enteritidis

Almost any food tainted by infected animal feces will carry *Salmonella* bacteria. Unpasteurized milk and eggs are especially high-risk carriers. Infections have leveled off, yet an emerging strain shows high rates of drug resistance.

Listeria monocytogenes

Able to survive refrigeration, *Listeria* is found in many foods, especially soft cheeses and processed meats like hot dogs. Pregnant women, newborns, and people with weakened immune systems are most susceptible.



Shigella sonnei

Contaminated water fouled by sewage or manure transfers *Shigella* to food. The bacteria cause a highly infectious disease that is spread by direct contact. Children in day-care settings are most vulnerable.

Chemical Dependency

TO IMPROVE YIELDS, FARMERS RELY ON PESTICIDES, GROWTH HORMONES, AND ANTIBIOTICS. WHAT'S GOOD FOR US, AND WHAT'S NOT?

The pesticides and drugs that underpin the mass production of food are sparking new concerns about how they affect our bodies. Pesticide residues exist on most of the non-organic produce we eat. Though government tests in the United States show that almost all residues fall within safety margins, alarm about the effects of pesticides on the development of children's nervous systems led to the recent banning of the popular pesticide methyl parathion for use on most fruits and vegetables. In developing nations such as India (top), its continued application threatens consumers, sprayers, and the environment.

Recombinant bovine growth hormone (rBGH) increases milk production in cows.

Canada has not approved its use. But dairy farmer Jim Mlsna of Hillsboro, Wisconsin (above), swears by rBGH. Injections every 14 days for his 300 cows increase daily milk production by ten pounds an animal. The FDA deems milk from hormone-treated cows safe, and Mlsna says: "I wouldn't let my children drink the farm milk if I didn't think it was safe."

The practice of administering antibiotics to food animals to speed growth has also raised health concerns. Food pathogens are apparently becoming resistant to antibiotics because of overexposure.

At Children's Hospital and Regional Medical Center in Seattle, microbiologist Joan Guzzo tests several antibiotics on a plate to see which ones will kill a sample of *E. coli* bacteria that has been isolated from a child's urinary tract.



greatest hazards today in the American food supply are not pesticide residues or dioxins or even hidden allergens but foodborne pathogens—bacteria, viruses, parasites—with the potential to harm or kill us.

Once, while on assignment for this magazine, I fell ill in an oceanside town in California after a hotel dinner of seafood and salad. That night I broke into a feverish sweat and suffered abdominal cramps and multiple bouts of diarrhea. By morning I was weak and depleted, but my symptoms were gone. If the statistics hold true, most Americans suffer in this way from time to time. We get a bad stomach for a brief spell or a bout of vomiting or diarrhea. These short-term ailments of our alimentary tracts are typically caused by viruses—often foodborne—and can spread from one person to the next by what is known as the fecal-oral route (contact with human waste and unwashed hands). The symptoms are usually mild and are gone in a day or two.

For some, however, experience with tainted food has another ending, no less chilling for its relative rarity.

A week before Christmas 1992 Lauren Beth Rudolph ate a cheeseburger from a Jack in the Box restaurant in California. On Christmas Eve, suffering from severe cramps and bloody diarrhea, Lauren was admitted to the hospital. There she endured three massive heart attacks and fell into a coma before dying on December 28. She was six years old.

The burger Lauren ate was contaminated with the virulent bacterium *Escherichia coli* O157:H7. Her death was what epidemiologists refer to as an index case, the first in an outbreak that caused 732 illnesses in five states and killed four children. Every year some 73,000 Americans become ill and 60—most of them children—die from *E. coli* O157:H7. The bug is a close cousin of the beneficial *E. coli* that normally reside in our own digestive systems. But so virulent is this version that it takes no more than a few organisms to cause deadly infection.

“We used to think of foodborne illness as little more than a stomachache,” says Joseph Levitt, director of the FDA’s Center for Food Safety and Applied Nutrition. “After the Jack in the Box incident we realized this was no issue of stomachaches, but a serious and compelling public health problem.”

THERE ARE 200 TIMES AS many bacteria in the colon of a single human as there are human beings who have ever lived. Most of these microbes coexist peacefully with our own cells and even assist them, helping with digestion, synthesizing vitamins, shaping the immune system, and fostering general health. Nearly all raw food, too, harbors bacteria. But the microbes that produce foodborne illness are bugs of a different order, capable of causing severe illness and even lasting damage—disorders ranging from temporary paralysis to kidney disease.

Many of these microbes are present in the animals we raise for food. When a food animal containing pathogens is slaughtered, its stomach contents or manure can taint meat during processing. Fruits and vegetables can pick up the pathogens if washed or irrigated with water contaminated with manure or human sewage.

And since a single bacterium, given the right conditions, divides rapidly enough to produce colonies of billions over the course of a day, even only lightly contaminated food can become highly infectious. The microbes can also hide and multiply on sponges, dish towels, cutting boards, sinks, knives, and countertops, where they’re easily transferred to food or hands.

A century ago typhoid fever, cholera, botulism, and trichinosis were common. The incidence of these diseases has fallen, at least in the developed world, thanks to improvements in food sanitation and safety—better animal husbandry, refrigeration, the pasteurization of milk, sophisticated canning and food preservation techniques. Consumers are better educated about how to clean and cook meats and produce, and standards put in place by the federal government have largely rid dining tables in the U.S. of food that is spoiled, contaminated with filth, or derived from sick animals.

But other foodborne infections have taken the place of the old ones—among them, a troubling cluster caused by bacteria with unwieldy names: *Campylobacter jejuni*, *Salmonella enteritidis*, *E. coli* O157:H7, *Shigella sonnei*, *Listeria monocytogenes*. Some of these are new forms of old microbes; others are the same as they’ve always been but are popping up in new places. The foods contaminated with this nasty set of pathogens tend to look, smell, and taste



normal, and the offending microbes, we are learning, can survive the traditional heating and cooling techniques we once thought did away with them.

GROWING UP IN THE 1960S, I remember the first thing I ever tasted that I wanted to taste again was cookie dough, a sweet, melting mix of butter, brown sugar, and raw eggs. I licked the dough bowl frequently over the years with no ill effect. The wisdom used to be that one should avoid only those raw eggs with cracked shells, which might allow pathogens in.

But now food experts agree that even a perfect egg may not be safe. *Salmonella enteritidis*—bacteria that can cause diarrhea, cramps, fever, and, in those with weakened immune systems, life-threatening infection—can get inside the ovaries of a laying hen and contaminate her eggs before the shells are formed. Beginning in September 2001 the FDA required that all egg cartons carry a safe-handling label stating: “To prevent illness from bacteria: keep eggs refrigerated, cook eggs until yolks are firm, and cook foods containing eggs thoroughly.” Soft-boiled eggs, eggs fried sunny-side up, and even softly scrambled eggs are now on the “unsafe” list along with classic versions of many egg-containing recipes touted in the cookbooks of the past few decades: Caesar salad, mousses, hollandaise sauce, French toast, omelettes, eggnog. It’s not wise to make mayonnaise or ice cream with eggs at home anymore either.

In my childhood I also ate with impunity the semi-raw hamburgers served at family picnics, undercooked either because of impatient

appetites or a fashion for rare meat. It’s clear now that *E. coli* O157:H7 can survive the gentle heating we give our rare hamburgers. The USDA advises cooks at home to heat ground beef until it’s no longer pink and reaches a temperature of 160°F. The standard for commercial food services, where equipment is more reliable, is 155°F.

Other potential troublemakers include pâtés, hot dogs, sliced deli meats, smoked fish, blue cheese, or soft cheeses such as Brie and Camembert because of the dangers of *Listeria*. This organism multiplies at refrigerator temperatures. In one study the microbe turned up on the inside surfaces of the refrigerators of two-thirds of the patients infected with *Listeria*. It doesn’t always get into our food, but when it does, it can cause encephalitis or meningitis in people with vulnerable immune systems and, in pregnant women, miscarriage or stillbirth.



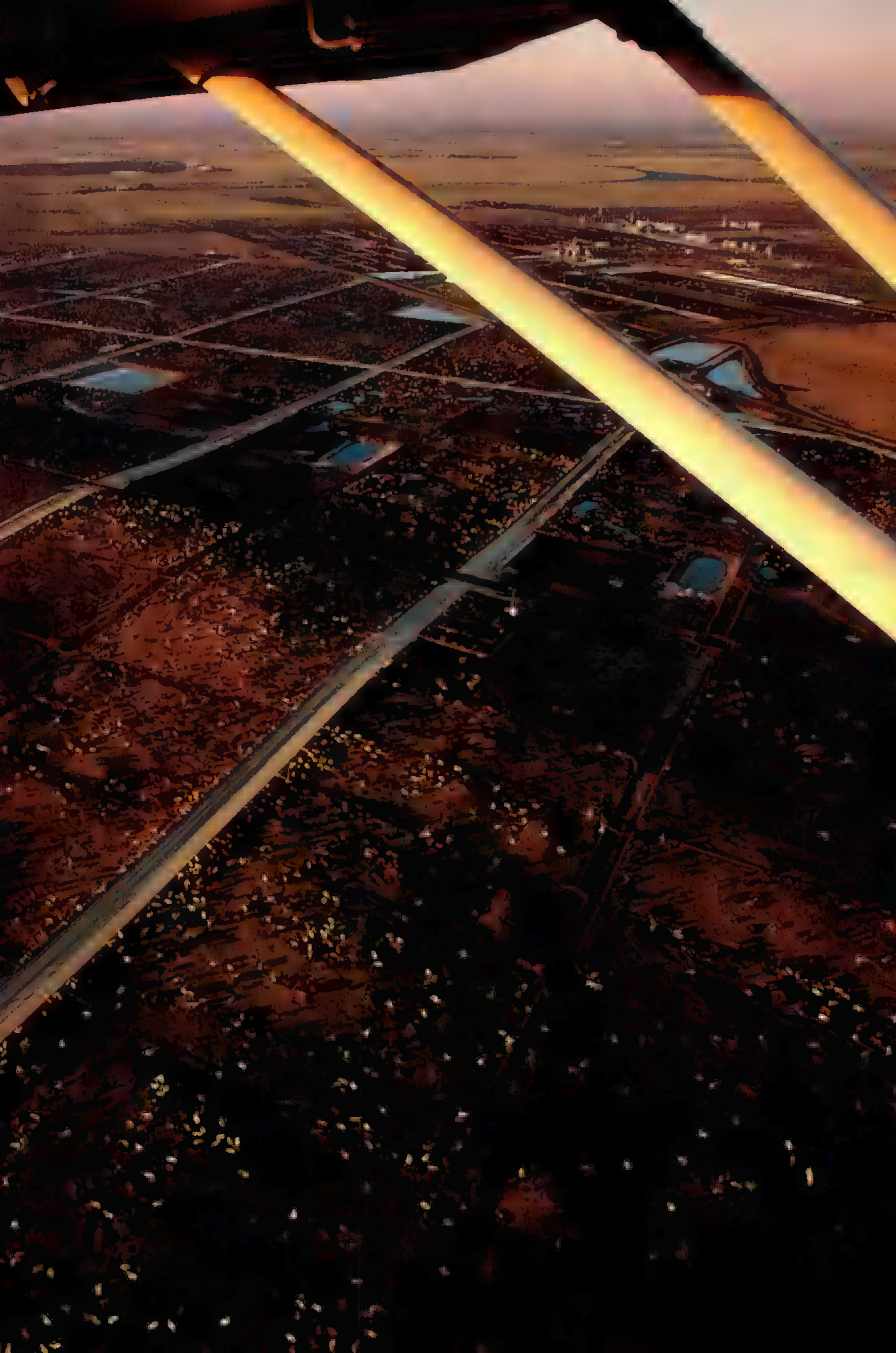
THE DANISH 1993

A 1993 food-poisoning outbreak in Denmark that sickened 700 people who had eaten pork tainted with *Salmonella* has led to a model cleanup of the industry. Before arriving at the Danish Crown slaughterhouse in Odense (left), all hogs are tattooed to allow the 50 plant veterinarians to trace the source of any problem. Egg producer Danæg combats *Salmonella* at its Christiansfeld plant by running eggs under ultraviolet rays (right) to kill surface bacteria.



A LOT OF BEEF

Penned between grain troughs, 100,000 cattle can fill this feedlot in Colorado, one of the biggest in the country. Large-scale agriculture keeps prices low but raises the risk of fecal contamination and disease.



“WHETHER THE overall incidence of foodborne disease has risen over the past generation is not known because we can’t track all foodborne illnesses,” says Patricia Griffin, chief of the CDC’s Foodborne Diseases Epidemiology Section. “What is clear is that the incidence is high, that some foodborne illnesses have clearly increased, and that dramatic changes in our food production system are likely to be playing a major role.”

Until the Jack in the Box outbreak, many

the CDC investigates outbreaks by comparing those who have become ill from food with closely matched individuals who are well and by comparing what the two groups ate. In this way they can identify both the food source of an outbreak and the contaminating microbe.

To link cases together, the scientists use a powerful new tool called PulseNet, a network of public health laboratories connected by computer that matches strains of microbes through DNA fingerprinting. PulseNet allows epidemiologists to associate an illness in Nebraska,



consumers believed that people got sick from food they didn’t cook right. “Now,” Griffin says, “we are more aware that the responsibility does not rest solely with the cook. We know that contamination often occurs early in the production process—at steps on the way from farm or field or fishing ground to market.”

Griffin has been in the foodborne-disease business for 15 years. Her job is to look for trends in food-related illness through analysis of outbreaks. Her staff of epidemiologists at

say, with one in Texas, tying together what might otherwise appear as a sea of sporadic and unrelated cases.

Then it’s the job of the investigators to track down the nature and mechanism of the contamination—what changed or went wrong in the food’s journey to the table—and to determine whether to recall a particular food or to change the process by which it’s produced.

In January 2000 public health officials in Virginia noted an unusual cluster of patients

sick with food poisoning from one strain of *Salmonella*. Using PulseNet, the CDC identified 79 patients in 13 states who suffered infection from the same strain of the microbe. Fifteen had been hospitalized with severe bloody diarrhea; two had died. The common factor? All had eaten mangoes during the previous November and December.

An investigation of the implicated fruit led to a single large mango farm in Brazil. When a team of health officials visited the farm, they discovered that tanks used to dip the mangoes in warm water to control fruit fly infestation, and then in cold water to cool the fruit, were open to the air. There were toads and birds around the tanks and feces in the water. It likely was the cold rinse that caused the mangoes to absorb the tank water and the pathogens it contained, including a strain of *Salmonella*.

It was a small problem, easily fixed. "That's one reason we investigate outbreaks," explains Griffin, "to find the little things that need to be corrected. These are not generally flukes; they're problems in the way food is produced. They're likely to have happened before and—if they're not fixed—will happen again." Since the mango incident, the farm in Brazil has enclosed its warm-water tanks, and the fruit is air-cooled with fans.

But the mango outbreak had a larger lesson: In the U.S. we no longer eat only fruits and vegetables in season and grown locally, as we once did. Instead, we demand our strawberries, peaches, mangoes, and lettuce year-round. As a result, we are depending more and more on imports. Over 40 percent of all fresh fruit consumed in the U.S. comes from Mexico, Chile,

Guatemala, Costa Rica, and other foreign countries, traveling hundreds, even thousands, of miles to reach our grocery-store shelves.

Eating food grown elsewhere in the world means depending on the soil, water, and sanitation conditions in those places and on the way their workers farm, harvest, process, and transport the products. (This is true, as well, for other nations that consume food exported from the U.S. Last spring, almonds from a farm in California infected 160 Canadians with *Salmonella*.) Because of the globalization of our food supply, the health hazards of one nation easily become those of another.

NOT ONLY DO WE LIKE OUR FOODS diverse and available year-round, but we also like them convenient—prepackaged, preferably, and ready-to-eat. This means that we're leaving to commercial foodmakers the peeling, chopping, and mixing of our food. We're buying lettuce in plastic packages and potato salad, tabbouleh, and hummus in deli containers. We're eating out more: Forty cents out of every U.S. dollar spent on food is spent outside the home in restaurants and other commercial food services, where young or inexperienced, and often underpaid, workers are preparing our meals. All of this raises our risks of food poisoning. "The more untrained people handling food, the greater the risk of inadequate cooking or of cross-contamination of safe foods from unsafe or uncooked foods," notes Griffin.

We also like our food cheap. No developed nation spends less of its wealth on meals than the U.S. Advances in technology spurred by pressure to keep food prices low have shifted

A Matter of Degrees

A street vendor goes with his instincts to know when his kabobs are ready to eat in Bangalore, India. Across the globe in a McDonald's restaurant in St. Charles, Illinois, the staff trusts only temperature readings. Floor manager Florencia Garcia jabs a pyrometer into quarter pounders held in a heating cabinet (right). McDonald's serves burgers cooked to 155°F. Conducting his daily corporate-mandated safety check, owner Anthony Lardas registers the numbers.



Every year some 73,000 Americans become ill and 60—most

the balance of food production in the U.S. from many small plants to fewer but larger ones. This magnifies the extent of harm that can arise from a single failure in food safety. In fact, some of the largest and most serious outbreaks of foodborne illness have resulted not from imported foods but from the factories and farms within our own borders, which provide food to huge numbers of consumers.

A 1994 case involving contaminated ice cream constitutes one of the largest outbreaks ever recorded. Trucks transporting the premix for Schwan's, a widely distributed brand of ice cream, carried traces of raw eggs contaminated with *Salmonella enteritidis*. The outbreak sickened an estimated 224,000 people in 48 states.

One of the deadliest outbreaks on record involved various brands of hot dogs and cold cuts made with meat from a Sara Lee processor. The microbe, an unusual strain of *Listeria*, sickened scores of consumers in 1998 and was linked to 15 deaths and 6 miscarriages or stillbirths. The outbreak ended after the company recalled 15 million pounds of meat—one of the largest meat recalls in U.S. history.

IN THE NAME OF EFFICIENCY and economy, we have also changed the way we raise our food animals. Our fish, cattle, and broiler and laying chickens are raised in giant "factory" farms, which house large numbers of animals in tight quarters. Griffin and others at the CDC worry that the conditions in these concentrated animal cities favor contamination and the spread of disease.

This is true in cattle feedlots, where animals are held in large groups for fattening before they're brought to slaughter. "Have you ever seen one of these places?" Griffin asks. "The cattle are jammed together, standing on black stuff, which is all feces. By the time they reach the slaughterhouse, they're covered with feces and crowded together. Even if only one animal is carrying *E. coli* O157:H7, under these conditions it will probably spread to others."

The Jack in the Box outbreak is a case in point. When beef is processed into ground beef, the chances of contamination rise significantly. Processing meat contaminated by one animal can spread the pathogen to all the hamburger that passes through the machinery in one day.

The USDA inspects every carcass in every meat and poultry processing plant—but without checking for microbial pathogens. "Meat inspectors still rely on sight, touch, and smell to spot disease," says Mike Taylor, former administrator of USDA's Food Safety and Inspection Service. "But the real problem in food, the bacteria that make people sick, can't be found that way."

In the summer of 1999, scientists from the USDA visited four large slaughterhouses in the Midwest to test beef cattle for *E. coli* O157:H7 contamination. The team found that 28 percent of the cattle entering slaughterhouses were infected and 43 percent of the skinned carcasses were contaminated. By the end of processing, however, only 2 percent of the tested meat was tainted, suggesting that measures taken by meat processors may be helping to reduce contamination.

One such measure, put in place in the past several years by food-processing companies, is a system called Hazard Analysis and Critical Control Point (HACCP) plan. This system, overseen by government regulatory agencies, scrutinizes threats to the safety of meat, poultry, seafood, and juice during processing. With HACCP, companies identify the key vulnerable points where contamination can occur in their slaughterhouses or processing plants. They must take steps to minimize the risk of pathogens invading at these points, after which they undergo government auditing.

Many U.S. meat processing plants also address contamination problems with procedures that include chemical baths, rinses, or sprays; bathing carcasses in steam; or irradiating processed meat to kill microbes. But while these measures have likely helped reduce the amount of contamination in meat products, it could be lower still, say scientists, if farmers reduced infection in their livestock. That's no easy task: *E. coli* O157:H7 is already widespread among cattle herds, and so far there's no proven, effective treatment. Researchers are exploring the possibility of using vaccines against the bug, treating livestock drinking water, even inoculating food animals with healthy gut bacteria to keep the pathogens out.

Patricia Griffin believes that finding *E. coli* in produce is an even more compelling argument

of them children—die from *E. coli* O157:H7.

for addressing the food-animal issue than finding it in meat. The problem arises for the most part from manure. “As a society,” notes Griffin, “we’re extremely careful with human feces. But cattle feces works its way into streams and groundwater, which we use to irrigate and wash our produce. Manure is also used as fertilizer. If it contains *E. coli* and *Salmonella*, we are recirculating these pathogens through our environment.

“At least you can kill *E. coli* in ground beef by cooking it at high enough temperatures,” she continues. “But these organisms stick to produce. By washing, you can reduce contamination, but if the pathogenic organisms are there, it’s unlikely that you’ll get them all off. What are you going to do, not eat lettuce?”

JUST HOW OUR FOOD ANIMALS are becoming infected with these pathogens in the first place remains a mystery. But the answer may lie in what *they* eat.

Farmers have in the past 50 years shifted the diets of beef cattle from hay to grain in order to boost growth rates and reduce costs. “When ruminants are fed fiber-deficient rations,” write USDA’s James B. Russell and Jennifer Rychlik of Cornell University, “microbial ecology is altered, and the animal becomes more susceptible to metabolic disorders and, in some cases, infectious diseases.”

In addition, new technologies have encouraged the feeding of a wider range of materials to cattle, including wastes. “Chickens in the U.S. eat a variety of feed, including fish meal from Asia,” explains Frederick Angulo of the CDC. “Cattle eat such agricultural by-products as peanut hulls, almond shells, waste from bakeries, and poultry manure. These commodities are shipped all over the world.”

By recirculating animal by-products and waste, we may be creating new niches and opportunities for foodborne pathogens to enter the food supply and spread. In Great Britain evidence of the dangers of using animal by-products in livestock feed surfaced in the outbreak of mad cow disease, or bovine spongiform encephalopathy (BSE). The rapid spread of the illness, which likely resulted from feeding cattle meat and bonemeal from animals that already had the disease, was linked with more

than a hundred cases of deadly Creutzfeldt-Jakob brain disease in humans who had consumed the infected meat. Since the outbreak among cattle in Great Britain in 1986, BSE has been found in animals in several European countries and Japan.

In 1997 the FDA banned the use of rendered remains of dead cattle and sheep in feed for U.S. ruminants, and there is no sign of BSE yet in the U.S. But many consumer groups are concerned that the government rules for animal feed include too many loopholes. Regulations still allow the use of animal blood and blood products as well as pig and horse protein. They also allow poultry to be used in cattle feed and cattle to be used in poultry feed. Is this an effective recycling of animal protein or a breach in a basic ecologic relationship—with serious consequences for our food supply?

Also present in the troughs of our food animals may be an even greater health hazard than pathogens themselves: antibiotics.

In the summer of 1998 a 62-year-old Danish woman was admitted to the emergency room at a hospital in Copenhagen after suffering diarrhea for nine days. She was diagnosed with foodborne *Salmonella* and immediately treated with ciprofloxacin, the antibiotic used to treat anthrax and one of the drugs of choice for *Salmonella* infection. But the drug did little good, and bacteria perforated her intestines. Surgery was unsuccessful, and the woman died of organ failure.

The *Salmonella* that infected the woman is known in the code language of epidemiologists as DT104. It is a relatively new strain that has evolved resistance to five antibiotics and sometimes shows resistance to those most commonly used to treat the infection it causes. Danish epidemiologists found the DT104 strain of *Salmonella* in 25 patients, all of whom had either eaten contaminated pork, handled it, or been exposed to someone sickened by it. The meat was traced through a slaughterhouse on the Danish Island of Zealand to two different swine herds. The pigs were carrying bacteria that had acquired resistance to the quinolone class of antibiotics. However, scientists were unable to determine how the pigs had become contaminated.

Farmers have been (Continued on page 30)



A Healthy Catch

UNLIKE STEAKS, FISH COME WITH NO INSPECTION SEAL. BUT FISH PRODUCERS MEET GOVERNMENT STANDARDS TO ENSURE SAFETY AND FRESHNESS.

Keeping fresh fish moving fast and keeping it chilled at all times are two important factors in eliminating pathogens from the plate.

Legal Sea Foods, a Boston-based restaurant chain, was an early leader in instituting modern handling practices. For company buyers each day begins at 5 a.m. at piers in Gloucester, New Bedford, and Boston. They look for fish caught during the previous 48 hours, like the haddock unloaded in Gloucester, Massachusetts, by fishermen Jose Batista and Manuel Bianco (left). Head buyer Bill Holler (top)

“We look at the eyes and the gills. We smell the fish. You want a sheen on the skin, a slime we call butter.”

—Roger Berkowitz, CEO, Legal Sea Foods

selects fish he will bid on at the morning auction. A big cod catches his eye because its body is still stiff, a sign of prime freshness.

The company eschews middlemen. Buyers send the fish in refrigerated trucks directly to the company’s quality-control laboratory and processing plant in the Boston suburb of Allston. There two safety procedures common throughout the industry are conducted. Microbiologists check fish and shellfish for toxins and bacteria, and other employees, like Jose Lainez and Matthew Drayton (middle) candle fish, which involves screening fillets on a light table to look for parasites. Then it’s back to the refrigerator for the final leg of the trip to one of Legal Sea Foods’ 26 U.S. restaurants.

Legal Sea Foods is proud of the series of safety checks it runs before the fish hits the cooking fire. They also hike menu prices to cover costs. But judging by the popularity of this high-end fish restaurant, many customers don’t mind.







HANDY REMINDER

Doris Welch, an Extension agent in Kearny County, Kansas, uses glow powder to demonstrate how bacteria can coat skin if people don't wash properly.



WITH OVER FORTY PERCENT OF OUR FRESH FRUIT COMING FROM ABROAD, FOREIGN GROWERS ARE LEARNING NEW HABITS.

Shoppers at the central market in Bangalore, India (left), don't think twice about buying produce that has been sitting in dirt contaminated by, among other things, animal refuse. Food safety standards barely exist in most of the developing world but are changing in response to the cravings of foreign markets. In Central America, in particular, farming and processing practices have been transformed to keep up with the all-season demand for fresh produce in the United States.

To ensure that imported food meets U.S. regulations, the FDA has established food safety outreach programs in some 30 countries.

Simple but vital changes, like regular hand washing and use of proper toilets by field-workers, have made all the difference.

—Carmela Velázquez

Costa Rica is one success story. In the central mountains Gerardo Mendez Fallas (top) packs blackberries straight into their containers. He will check the fruit for debris at his home, and within 24 hours a plane will deliver the berries to Miami.

At the Adapex shipping facility near Cartago (middle) workers wear sanitary gear to pack mini-vegetables. All the produce has been rinsed in chlorinated water. Pineapples receive similar fastidious care at the Coopeagrinar plant in Zarcero (bottom).

Carmela Velázquez, a food scientist from the Universidad de Costa Rica who is integrally involved with the food safety program, believes that changes already made in her country bode well for the future. "The farmers we've trained," she says, "will become models for all our growers."



There are 200 times as many bacteria in the colon of a single

(Continued from page 23) adding antibiotics to animal feed for more than half a century, after it was discovered that the drugs were effective in accelerating the growth of animals. Now by some estimates the volume of antibiotics used in animal feed equals or exceeds that used in human medicine.

"The promiscuous use of antibiotics as food supplements for farm animals is a serious threat to human health," says CDC's Alicia Anderson, an epidemiologist for the National Antimicrobial Resistance Monitoring System (NARMS). Anderson and others believe that use of the drugs in healthy animals is playing a role in changing the very nature of foodborne bacteria, creating strains that are resistant to antibiotics used in human medicine.

Since the early 1990s infections with the "superbug" DT104 and other foodborne antibiotic-resistant bacteria have turned up in several countries. A report published in 2001 after scientists at the University of Maryland and the FDA sampled ground beef, turkey, chicken, and pork from supermarkets in Washington, D.C., revealed that a fifth of the samples contained *Salmonella*, and 84 percent of these organisms were resistant to at least one kind of antibiotic. Some were resistant to as many as 12.

Officials in the animal drug industry argue that antibiotics are critical for keeping food animals healthy, and experts agree that overuse of antibiotics among people, not animals, is the true cause of antibiotic resistance in humans. But many scientists say that dosing animals with the same antibiotics we rely on in human medicine is a bad idea. The World Health Organization has advised against the practice, and in 1999 the European Union prohibited four antibiotics used to treat human illnesses from use as livestock growth promoters.

Three of those four antibiotics are still used to treat human illness and to promote livestock growth in the U.S. And at least 13 more are approved for both uses. "We're out of sync with the rest of the developed world in how antibiotics are used," says Anderson.

There are, however, signs of change. In early 2002, three poultry companies announced that they had greatly reduced their use of antibiotics in healthy chickens. Consumer groups

and public health officials are hoping that other companies in the poultry, pork, and beef industries will follow their lead.

GOVERNMENT OFFICIALS and industry representatives are fond of boasting that despite the many threats we have discovered, Americans have the safest food supply in the world. This may or may not be true.

There are indications that since 1996 infections from *Campylobacter*, *Salmonella*, and *Listeria* have dropped slightly—perhaps as a result of consumer awareness combined with new programs in government and industry to ensure the safety of meat, eggs, juice, and fresh produce. Recently government agencies and the food industry initiated a food safety curriculum for students and a "Fight BAC!" campaign to inform consumers how to keep foods safe from harmful bacteria by cleaning them, separating them, cooking them to proper temperatures, and chilling them promptly. And the FDA has launched a training program called Good Agricultural Practices to help other nations train agricultural workers and food producers in safe methods of farming, harvesting, and producing food.

But critics of the nation's food safety net say that gaps created by antiquated, inconsistent laws and regulations as well as fragmented oversight are still allowing too many pathogens to slip into our food supply. Since 1996 more people have gotten sick from *Shigella*, and the number of deadly *E. coli* infections has held steady. FDA studies completed in 2001 reported finding either *Salmonella* or *Shigella* in 12 out of 1,028 samples of domestic fresh produce, from cantaloupes to scallions, cilantro to celery to lettuce. According to the CDC contaminated eggs caused 82 percent of the cases of *Salmonella enteritidis* between 1985 and 1998. A study by the Consumers Union a few years ago reported that two-thirds of chickens in U.S. grocery stores carry *Campylobacter*, bacteria that live in the intestines of healthy birds. When the bacteria survive because of improper cooking of chickens or when cross-contamination occurs in the kitchen, they can cause abdominal pain, fever, diarrhea, and vomiting in humans.

human as there are human beings who have ever lived.

Food is not sterile, and it cannot be made risk-free. But I'm asking myself, Do I accept some level of contamination as inevitable and increase my vigilance in the kitchen? Or do I insist on cleaner meat, poultry, eggs, and vegetables? And how far would I go to get them?

The Swedish chicken industry virtually eliminated *Salmonella* from their flocks by cleaning up their chicken houses and heating feed to rid it of the pathogen. Now Swedes buy *Salmonella*-free chickens. Are American consumers willing to pay extra for safer food of higher quality? "Many food producers say no," says Frederick Angulo, director of CDC's NARMS. "But do Americans really know that 10 percent of chickens have *Salmonella* and 60 to 80 percent have *Campylobacter*? And that 20 percent of *Campylobacter* is resistant to fluoroquinolones—the antibiotics of choice for treatment of *Campylobacter* infections?" he asks. Consumers may understand that cooking chicken properly will kill pathogens, but if they don't know how prevalent those pathogens are in the first place, can they make an informed choice about what to pay for?

Safe food is a moving target—in part because we are moving targets. Our eating habits and our ways of producing food change. We change. In this country the number of people most vulnerable to foodborne disease is growing. Within the next three decades a fifth of the population will be over 65, and many of them will be particularly susceptible to serious infection from *Salmonella*, *Listeria*, *E. coli*. Young children are more likely to be exposed to these bugs than they were a generation ago, not

only because the production of food has changed but also because families eat out or take home prepared food more often.

And the microbes themselves are changing, evolving, taking hold in new populations, through new food vehicles, causing more or new disease. We still have limited understanding of how these foodborne pathogens work. After nearly 20 years of research, we still can't consistently treat advanced *E. coli* O157:H7 infections. We still are searching for clues to how food pathogens spread among cattle, egg-laying hens, and broiler chickens.

How can we make safer the food and water that animals consume? How can we dispose of animal manure without threatening the environment and the food supply? How can we ensure the safety of imported foods and foods handled in our restaurants and kitchens?

These are some of the big questions that still need to be addressed to minimize our risk of infection from foodborne disease. In the meantime I am cooking my egg yolks thoroughly, washing my hands and countertops to avoid cross-contamination, and forbidding my children pink hamburger. And I am considering what choices we may have in the future about the safety of our own food, and how much extra I might be willing to pay to have my chicken guaranteed free of *Campylobacter*—or my salad free of deadly *E. coli*. □

ON WEBSITE

What's safe, what's not? Hear from photographer Jim Richardson in our award-winning Sights & Sounds series ■ nationalgeographic.com/ngm/0205. AOL Keyword: NatGeoMag

Beyond a Stomachache

Cheeseburgers nearly killed Brianne Kiner, middle. After eating undercooked beef infected with *E. coli* O157:H7 in 1993, the Seattle girl lay in a coma for six weeks. The meat was traced to a Jack in the Box restaurant. Still recovering, Brianne joins her mother, Suzanne, and lawyer William Marler to study a picture from one of her surgeries. Kiner won a 15.6-million-dollar settlement. Such costly corporate lessons—and vigilance by consumers—promise to make our food safer to swallow.





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How Altered?

Scientists continue to find new ways to insert genes for specific traits into plant and animal DNA. A field of promise—and a subject of debate—genetic engineering is changing the food we eat and the world we live in.

In the brave new world of genetic engineering, Dean DellaPenna envisions this cornucopia: tomatoes and broccoli bursting with cancer-fighting chemicals and vitamin-enhanced crops of rice, sweet potatoes, and cassava to help nourish the poor. He sees wheat, soy, and peanuts free of allergens; bananas that deliver vaccines; and vegetable oils so loaded with therapeutic ingredients that doctors “prescribe” them for patients at risk for cancer and heart disease. A plant biochemist at Michigan State University, DellaPenna believes that genetically engineered foods are the key to the next wave of advances in agriculture and health.

While DellaPenna and many others see great potential in the products of this new biotechnology, some see uncertainty, even danger. Critics fear that genetically engineered products are being rushed to market before their effects are fully understood. Anxiety has been fueled by reports of taco shells contaminated with genetically engineered corn not approved for human consumption; the

BEHOLD THE FUTURE OF THE APPLE

If the gene inserted into this apple plantlet makes it resistant to the fire blight bacterium, it could help save apple growers tens of millions of dollars a year. Researchers are also working on an apple that could vaccinate children against a virus that is the leading cause of childhood pneumonia.



potential spread of noxious “superweeds” spawned by genes picked up from engineered crops; and possible harmful effects of biotech corn pollen on monarch butterflies.

In North America and Europe the value and impact of genetically engineered food crops have become subjects of intense debate, provoking reactions from unbridled optimism to fervent political opposition.

Just what are genetically engineered foods, and who is eating them? What do we know about their benefits—and their risks? What effect might engineered plants have on the environment and on agricultural practices around the world? Can they help feed and preserve the health of the Earth’s burgeoning population?

Q: Who’s eating biotech foods?

A: In all likelihood, you are.

Most people in the United States don’t realize that they’ve been eating genetically engineered foods since the mid-1990s. More than 60 percent of all processed foods on U.S. supermarket shelves—including pizza, chips, cookies, ice cream, salad dressing, corn syrup, and baking powder—contain ingredients from engineered soybeans, corn, or canola.

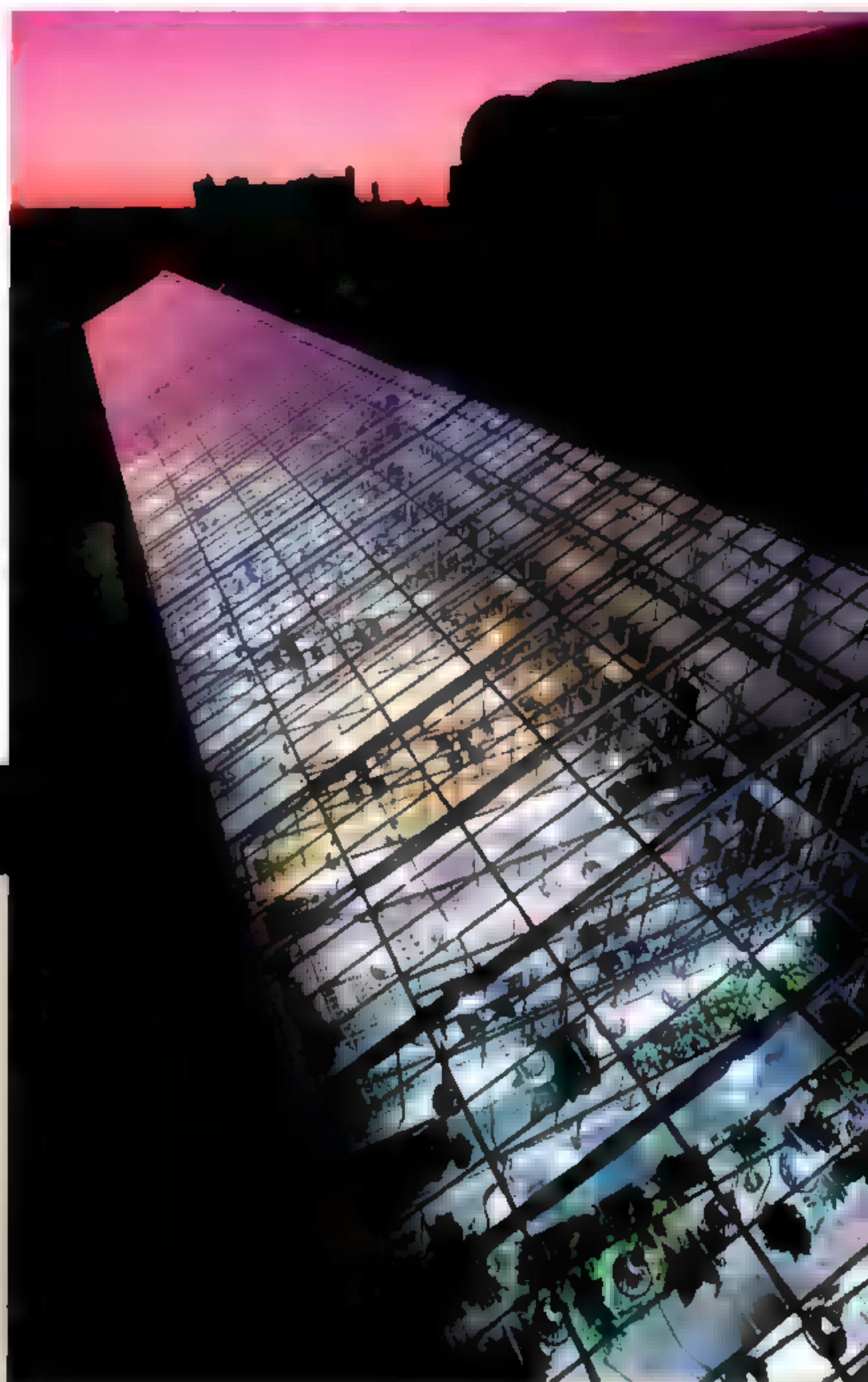
In the past decade or so, the biotech plants that go into these processed foods have leaped from hothouse oddities to crops planted on a massive scale—on 130 million acres in 13 countries, among them Argentina, Canada, China, South Africa, Australia, Germany, and Spain. On U.S. farmland, acreage planted with genetically engineered crops jumped nearly 25-fold from 3.6 million acres in 1996 to 88.2 million acres in 2001. More than 50 different “designer” crops have passed through a federal

review process, and about a hundred more are undergoing field trials.

Q: How long have we been genetically altering our food?

A: Longer than you think.

Genetic modification is not novel. Humans have been altering the genetic makeup of plants for millennia, keeping seeds from the best crops and planting them in following years, breeding and crossbreeding varieties to make them taste sweeter, grow bigger, last longer. In this way we’ve transformed the wild tomato, *Lycopersicon*, from a fruit the size of a marble to today’s giant, juicy beefsteaks. From a weedy plant called teosinte with an “ear” barely an inch long has come our foot-long ears of sweet white and yellow corn. In just the past few decades plant breeders have used traditional techniques to produce varieties of wheat and rice plants with higher grain yields. (Continued on page 41)



From Hothouse to Built

At one of Cornell University’s massive greenhouses, researcher Ajay Garg examines rice plants engineered to tolerate conditions that would kill ordinary rice. Inserting a pair of bacterium genes into rice DNA produces trehalose, a sugar that keeps cells from disintegrating under stresses such as drought, salt, and cold, which limit rice production worldwide. With around 30 percent of the world’s calories coming from rice, hardier varieties could help feed a growing population.

FROM THE LAB TO YOUR DINNER PLATE

To the eye and palate, most biotech foods on the market are indistinguishable from their conventional counterparts. More than 70 percent of today's engineered crops have been developed for herbicide resistance, and most of the rest are engineered to contain a gene from the *Bacillus thuringiensis* (Bt) bacterium, which produces a natural insecticide. Corn has been modified for both. These products and others, such as fast-growing salmon (not yet on the market), benefit mostly producers, but crops to benefit consumers, such as "golden rice," a possible tool to fight vitamin A deficiency, are in the works.





Pros and Cons

BIOTECHNOLOGY HAS BENEFITS AND RISKS, BOTH ACTUAL AND POTENTIAL.

Proponents and detractors of genetic engineering agree on at least one thing: Biotechnology is in its infancy. Proponents say the gains that now accrue mainly to biotech companies and big-business farmers will spread as consumers get more nutritious foods, and farmers in the developing world get healthier and higher yield crops. Detractors say genetic engineering is a Pandora's box, and we are releasing uncontrollable forces into our environment and food supply. Recently in Mexico, birthplace of corn and storehouse of its genetic diversity, researchers reported that local varieties had been contaminated by modified genes—even though Mexico has banned the planting of engineered corn. Some scientists question the findings of the report, but others see them as evidence of risks.

BENEFITS

Higher Yields Engineered crops can help feed the overpopulated world, where poor farming conditions and low-tech practices leave yields far below the potential.

Fewer Pesticides For the crop, crops, built-in insecticide has cut spraying dramatically. Herbicide-resistant plants can contribute to soil conservation.

Better Nutrition Staple foods that are low in protein or vitamins might be enhanced for both; other foods could have allergens or natural toxins removed.

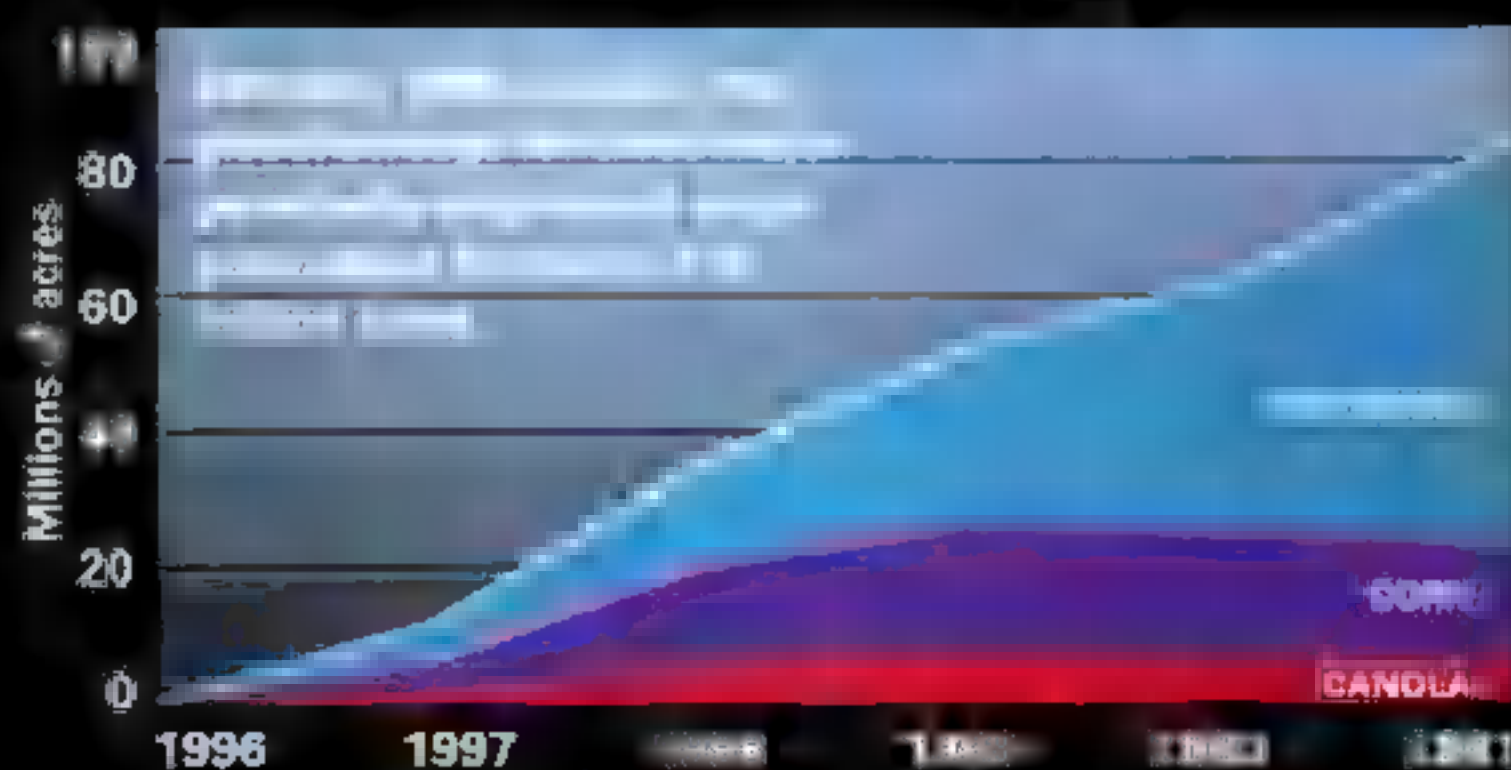
RISKS

Gene Flow Modified crops can spread their novel genes to wild relatives, and such altered organisms might become hard to manage.

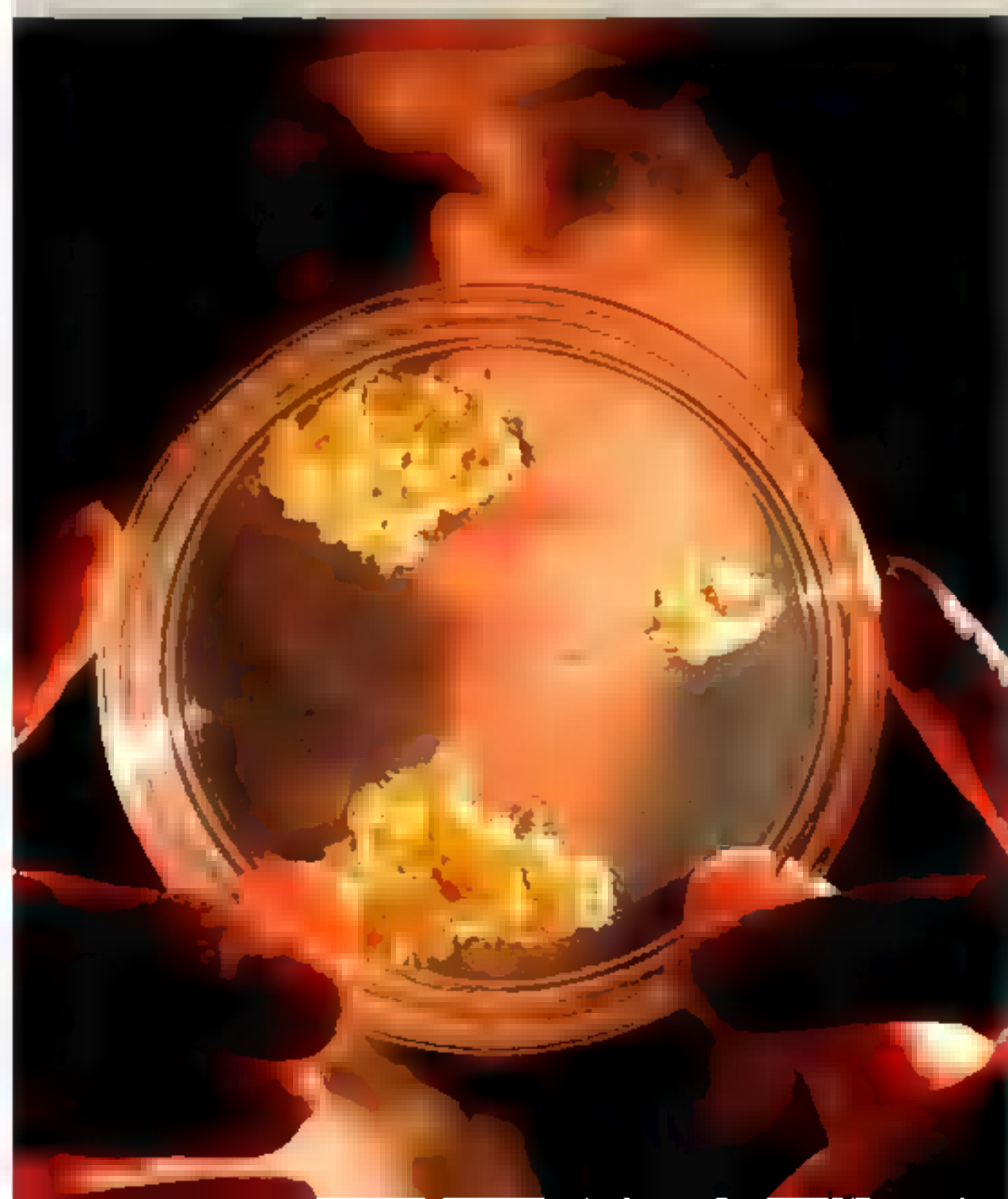
Collateral Damage Engineered crops may hasten insects' resistance to bio toxins. Toxin buildup in soil might have a negative effect on soil ecosystems.

Health Effects Allergens might be introduced into foods.

GENETICALLY ENGINEERED FOOD CROPS



ART BY TIM O'BRIEN; GRAPHIC BY TIMOTHY ALT. DATA SOURCES: THE INTERNATIONAL SERVICE FOR THE ACQUISITION OF AGRI-BIOTECH APPLICATIONS.



Genetic Engineering 101

How hard is it to change the building blocks of life? "I can take the next ten people off the street and have them making DNA in short order," says Ted Thannhauser, director of Cornell University's BioResource Center. Using a machine called a DNA synthesizer (top left), a technician at the center assembles different combinations of the four nucleotides—abbreviated as A, C, T, and G—that make up a gene. The order of the combination determines the traits that the gene will express.

There are several ways to introduce selected genes into the chromosomes of plants. One method involves inserting the gene into a soil bacterium called *Agrobacterium*, which



is then introduced to the target organism, bringing along part of its own DNA as well as the added gene.

A more versatile method is a device known as a gene gun, which was invented by a scientist who started out using a BB gun prototype to shoot genes into living cells. "People would shake their heads when they heard the idea," says Cornell researcher Bruce Reisch. "They'd chuckle, 'How's that ever going to work?'" But it did, and now researchers at Cornell and elsewhere use a more sophisticated version (left center) to propel tiny particles of gene-coated tungsten or gold directly into cells. Once that's done, scientists watch for cell

growth to see which cells have successfully incorporated the new genes. Out of every ten rice plants Cornell scientists modify, only one or two will be suited for further development. Researchers must monitor scores of specimen dishes (above) to determine which plants will offer improvements over the original rice.

The difference between a successful and unsuccessful modification can be as plain to the eye as corn-cell clusters in a petri dish (left). The clusters on the left have a gene that makes them resistant to a herbicide in the dish; the cluster on the right does not. Only the resistant cells are growing.



(Continued from page 35) They have also created hundreds of new crop variants using irradiation and mutagenic chemicals.

But the technique of genetic engineering is new, and quite different from conventional breeding. Traditional breeders cross related organisms whose genetic makeups are similar. In so doing, they transfer tens of thousands of genes. By contrast, today's genetic engineers can transfer just a few genes at a time between species that are distantly related or not related at all.

Genetic engineers can pull a desired gene from virtually any living organism and insert it into virtually any other organism. They can put a rat gene into lettuce to make a plant that produces vitamin C or splice genes from the cecropia moth into apple plants, offering protection from fire blight, a bacterial disease that damages apples and pears. The purpose is the same: to insert a gene or genes from a donor organism carrying a desired trait into an organism that does not have the trait.

The engineered organisms scientists produce by transferring genes between species are called transgenic. Several dozen transgenic food crops are currently on the market, among them varieties of corn, squash, canola, soybeans, and cotton, from which cottonseed oil is produced. Most of these crops are engineered to help farmers deal with age-old agriculture problems: weeds, insects, and disease.

Farmers spray herbicides to kill weeds. Biotech crops can carry special "tolerance" genes that help them withstand the spraying of chemicals that kill nearly every other kind of plant. Some biotech varieties make their own insecticide, thanks to a gene borrowed from a common soil bacterium, *Bacillus thuringiensis*, or Bt for short.

The Farmer's New Friends

At a farm-industry show in Nebraska, farmers comb the tracks of combines to see how the state-of-the-art machine fared on a state-of-the-art crop: Roundup Ready corn, a Monsanto product engineered to withstand its popular herbicide. Coming up for Monsanto: a Bt corn that resists the corn rootworm, a pest that costs U.S. corn farmers a billion dollars a year and accounts for more than half their insecticide use. At a Colorado test field (right) the new BT corn outperforms its conventional kin.

Bt genes code for toxins considered to be harmless to humans but lethal to certain insects, including the European corn borer, an insect that tunnels into cornstalks and ears, making it a bane of corn farmers. So effective is Bt that organic farmers have used it as a natural insecticide for decades, albeit sparingly. Corn borer caterpillars bite into the leaves, stems, or kernels of a Bt corn plant, the toxin attacks their digestive tracts, and they die within a few days.

Other food plants—squash and papaya, for instance—have been genetically engineered to resist diseases. Lately scientists have been experimenting with potatoes, modifying them with genes of bees and moths to protect the crops from potato blight fungus, and grapevines with silkworm genes to make the vines resistant to Pierce's disease, spread by insects.

With the new tools of genetic engineering, scientists have also created transgenic animals.



Atlantic salmon grow more slowly during the winter, but engineered salmon, “souped-up” with modified growth-hormone genes from other fish, reach market size in about half the normal time. Scientists are also using biotechnology to insert genes into cows and sheep so that the animals will produce pharmaceuticals in their milk. None of these transgenic animals have yet entered the market.

Q: Are biotech foods safe for humans?

A: Yes, as far as we know.

“Risks exist everywhere in our food supply,” points out Dean DellaPenna. “About a hundred people die each year from peanut allergies. With genetically engineered foods we minimize risks by doing rigorous testing.”

According to Eric Sachs, a spokesman for Monsanto, a leading developer of biotech products: “Transgenic products go through more testing than any of the other foods we eat. We screen for potential toxins and allergens. We monitor the levels of nutrients, proteins, and other components to see that the transgenic plants are substantially equivalent to traditional plants.”

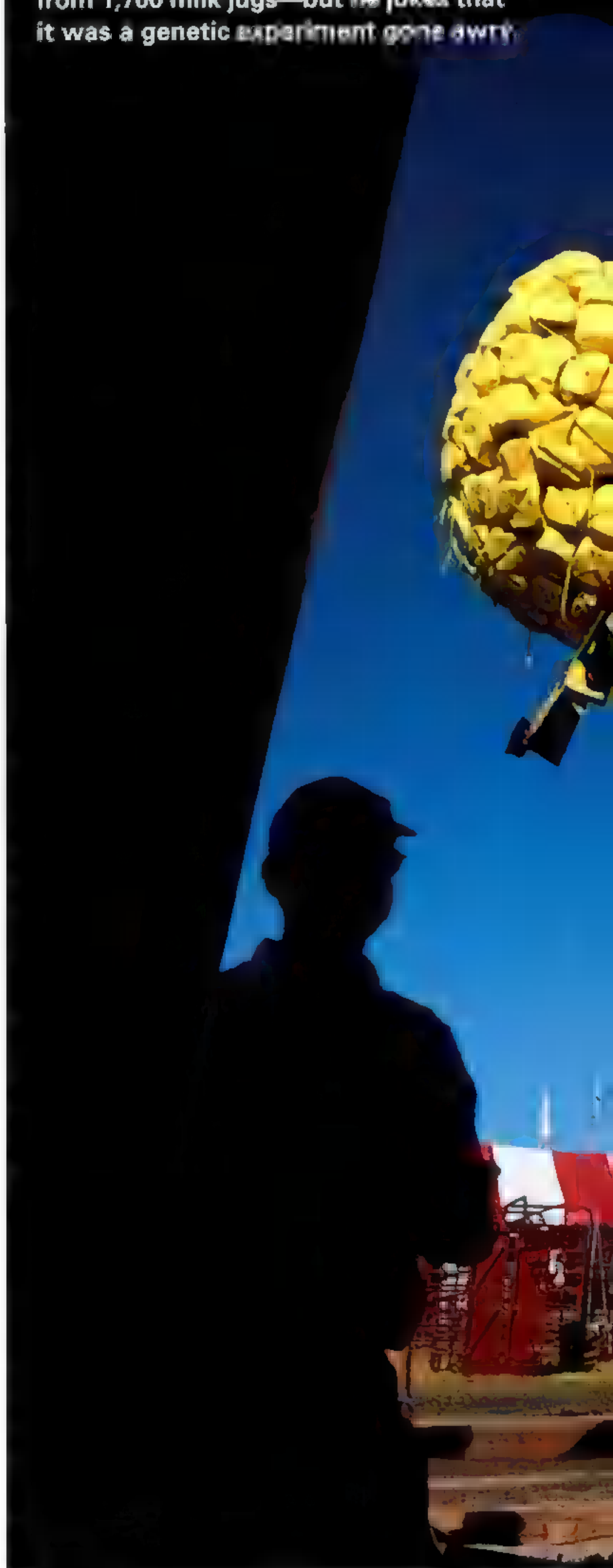
Three federal agencies regulate genetically engineered crops and foods—the U.S. Department of Agriculture (USDA), the Environmental Protection Agency (EPA), and the Food and Drug Administration (FDA). The FDA reviews data on allergens, toxicity, and nutrient levels voluntarily submitted by companies. If that information shows that the new foods are not substantially equivalent to conventional ones, the foods must undergo further testing. Last year the agency proposed tightening its scrutiny of engineered foods, making the safety assessments mandatory rather than voluntary.

“When it comes to addressing concerns about health issues, industry is being held to very high standards,” says DellaPenna, “and it’s doing its best to meet them in reasonable and rigorous fashion.”

In the mid-1990s a biotech company launched a project to insert a gene from the Brazil nut into a soybean. The Brazil nut gene selected makes a protein rich in one essential amino acid. The aim was to create a more nutritious soybean for use in animal feed. Because the Brazil nut is known to contain an allergen, the company also tested the product for human reaction, with the thought that the transgenic

CORNUCOPIA

A crowd favorite at corn-belt festivals, Dale Lingar’s giant ear of corn is made from 1,700 milk jugs—but he jokes that it was a genetic experiment gone awry.





EAR OF CORN © DALE UNGERER

soybean might accidentally enter the human food supply. When tests showed that humans would react to the modified soybeans, the project was abandoned.

For some people this was good evidence that the system of testing genetically engineered foods works. But for some scientists and consumer groups, it raised the specter of allergens or other hazards that might slip through the safety net. Scientists know that some proteins, such as the one in the Brazil nut, can cause allergic reactions in humans, and they know how to test for these allergenic proteins. But the possibility exists that a novel protein with allergenic properties might turn up in an engineered food—just as it might in a new food produced by conventional means—and go undetected. Furthermore, critics say, the technique of moving genes across dramatically different species increases the likelihood of something going awry—either in the function of the inserted gene or in the function of the host DNA—raising the possibility of unanticipated health effects.

An allergy scare in 2000 centered around StarLink, a variety of genetically engineered corn approved by the U.S. government only for animal use because it showed some suspicious qualities, among them a tendency to break down slowly during digestion, a known characteristic of allergens. When StarLink found its way into taco shells, corn chips, and other foods, massive and costly recalls were launched to try to remove the corn from the food supply.

No cases of allergic response have been pinned to StarLink. In fact, according to Steve L. Taylor, chair of the Department of Food Science and Technology at the University of Nebraska, “None of the current biotech products have been implicated in allergic reactions or any other healthcare problem in people.” Nevertheless, all new foods may present new risks. Only rigorous testing can minimize those risks.

Often overlooked in the debate about the health effects of these foods is one possible health benefit: Under some conditions corn genetically engineered for insect resistance may enhance safety for human and animal consumption. Corn damaged by insects often contains high levels of fumonisins, toxins made by fungi that are carried on the backs of insects and that grow in the wounds of the

damaged corn. Lab tests have linked fumonisins with cancer in animals, and they may be potentially cancer-causing to humans. Among people who consume a lot of corn—in certain parts of South Africa, China, and Italy, for instance—there are high rates of esophageal cancer, which scientists associate with fumonisins. Studies show that most Bt corn has lower levels of fumonisins than conventional corn damaged by insects.

Should genetically engineered foods be labeled? Surveys suggest that most Americans would say yes (although they wouldn't want to pay more for the labeling). Professor Marion Nestle, chair of the Department of Nutrition and Food Studies at New York University, favors labeling because she believes consumers want to know and have the right to choose. However, no engineered foods currently carry labels in the U.S. because the FDA has not found any of them to be substantially different from their conventional counterparts. Industry representatives argue that labeling engineered



foods that are not substantially different would arouse unwarranted suspicion.

Q: Can biotech foods harm the environment?

A: It depends on whom you ask.

Most scientists agree: The main safety issues of genetically engineered crops involve not people but the environment. "We've let the cat out of the bag before we have real data, and there's no calling it back," says Allison Snow, a plant ecologist at Ohio State University.

Snow is known for her research on "gene flow," the movement of genes via pollen and seeds from one population of plants to another, and she and some other environmental scientists worry that genetically engineered crops are being developed too quickly and released on millions of acres of farmland before they've been adequately tested for their possible long-term ecological impact.

Advocates of genetically engineered crops argue that the plants offer an environmentally friendly alternative to pesticides, which tend to pollute surface and groundwater and harm wildlife. The use of Bt varieties has dramatically reduced the amount of pesticide applied to cotton crops. But the effects of genetic engineering on pesticide use with more widely grown crops are less clear-cut.

What might be the effect of these engineered plants on so-called nontarget organisms, the creatures that visit them? Concerns that crops with built-in insecticides might damage wildlife were inflamed in 1999 by the report of a study suggesting that Bt corn pollen harmed monarch butterfly caterpillars.

Monarch caterpillars don't feed on corn pollen, but they do feed on the leaves of milkweed

plants, which often grow in and around cornfields. Entomologists at Cornell University showed that in the laboratory Bt corn pollen dusted onto milkweed leaves stunted or killed some of the monarch caterpillars that ate the leaves. For some environmental activists this was confirmation that genetically engineered crops were dangerous to wildlife. But follow-up studies in the field, reported last fall, indicate that pollen densities from Bt corn rarely reach damaging levels on milkweed, even when monarchs are feeding on plants within a cornfield.

"The chances of a caterpillar finding Bt pollen doses as high as those in the Cornell study are negligible," says Rick Hellmich, an entomologist with the Agricultural Research Service and one author of the follow-up report. "Butterflies are safer in a Bt cornfield than they are in a conventional cornfield, when they're subjected to chemical pesticides that kill not just caterpillars but most insects in the field."

Perhaps a bigger concern has to do with insect evolution. Crops that continuously make Bt may hasten the evolution of insects impervious to the pesticide. Such a breed of insect, by becoming resistant to Bt, would rob many farmers of one of their safest, most environmentally friendly tools for fighting the pests.

To delay the evolution of resistant insects, U.S. government regulators, working with biotech companies, have devised special measures for farmers who grow Bt crops. Farmers must plant a moat or "refuge" of conventional crops near their engineered crops. The idea is to prevent two resistant bugs from mating. The few insects that emerge from Bt fields resistant to the insecticide would mate with their nonresistant neighbors living on conventional crops

Biotech Beneficiaries

Orlando Manuel (left) is one of many papaya growers in Hawaii who owe their livelihood to genetic engineering. The papaya ringspot virus had the industry on the ropes until 1998, when farmers were given seeds modified for resistance. At Purdue University (right) researchers have bred "kinder, gentler" chickens and are trying to identify the specific genes responsible for the good behavior. Once they do, hogs might be next; aggression costs the hog industry billions.





nearby; the result could be offspring susceptible to Bt. The theory is that if growers follow requirements, it will take longer for insects to develop resistance.

It was difficult initially to convince farmers who had struggled to keep European corn borers off their crops to let the insects live and eat part of their acreage to combat resistance. But a 2001 survey by major agricultural biotech companies found that almost 90 percent of U.S. farmers complied with the requirements.

Many ecologists believe that the most damaging environmental impact of biotech crops may be gene flow. Could transgenes that confer resistance to insects, disease, or harsh growing conditions give weeds a competitive advantage, allowing them to grow rampantly?

“Genes flow from crops to weeds all the time when pollen is transported by wind, bees, and other pollinators,” says Allison Snow. “There’s no doubt that transgenes will jump from engineered crops into nearby relatives.” But since gene flow usually takes place only between closely related species, and since most major U.S. crops don’t have close relatives growing nearby, it’s extremely unlikely that gene flow will occur to create problem weeds.

Still, Snow says, “even a very low probability event could occur when you’re talking about thousands of acres planted with food crops.” And in developing countries, where staple crops are more frequently planted near wild relatives, the risk of transgenes escaping is higher. While no known superweeds have yet emerged, Snow thinks it may just be a matter of time.

Given the risks, many ecologists believe that industry should step up the extent and rigor of its testing and governments should strengthen

their regulatory regimes to more fully address environmental effects. “Every transgenic organism brings with it a different set of potential risks and benefits,” says Snow. “Each needs to be evaluated on a case-by-case basis. But right now only one percent of USDA biotech research money goes to risk assessment.”

Q: Can biotech foods help feed the world?

A: There are obstacles to overcome.

“Eight hundred million people on this planet are malnourished,” says Channapatna Prakash, a native of India and an agricultural scientist at the Center for Plant Biotechnology Research at Tuskegee University, “and the number continues to grow.”

Genetic engineering can help address the urgent problems of food shortage and hunger, say Prakash and many other scientists. It can increase crop yields, offer crop varieties that resist pests and disease, and provide ways to grow crops on land that would otherwise not support farming because of drought conditions, depleted soils, or soils plagued by excess salt or high levels of aluminum and iron. “This technology is extremely versatile,” Prakash explains, “and it’s easy for farmers to use because it’s built into the seed. The farmers just plant the seeds, and the seeds bring new features in the plants.”

Some critics of genetic engineering argue that the solution to hunger and malnutrition lies in redistributing existing food supplies. Others believe that the ownership by big multinational companies of key biotechnology methods and genetic information is crippling public-sector efforts to use this technology to address the needs of subsistence farmers. The

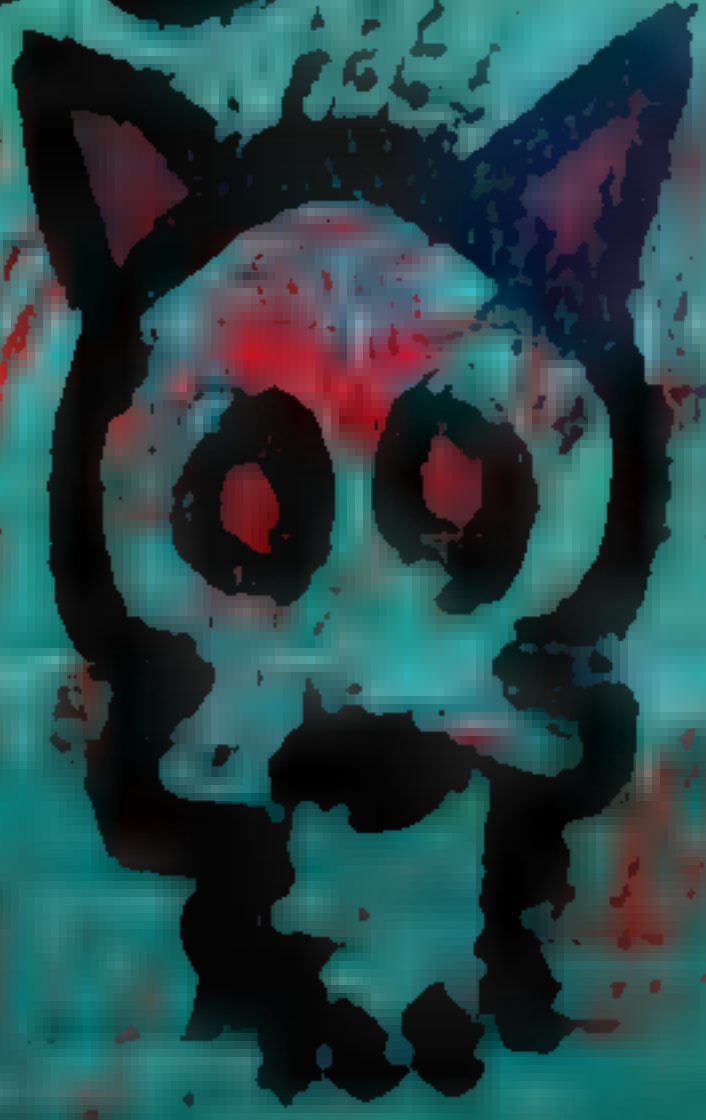
Unintended Consequences

Critics saw their fears realized in 2000, when StarLink corn, approved only for animal use, made it into stores. The corn, feared allergenic, was taken off the market, but grain elevators still test for contamination (left). A rare variety of Bt corn proved toxic in the lab to monarch caterpillars, which feed on milkweed in and around cornfields. Caterpillars that eat leaves dusted with this corn’s pollen are stunted or killed (left petri); a variety with less Bt toxin in its pollen does no apparent harm.





MONSATAN



The Battle Over Food

AS NEW PRODUCTS HIT THE MARKET, THE BIOTECH INDUSTRY GATHERED OPPOSITION ALMOST AS FAST AS IT DID STEAM.

For some opponents of genetic engineering, Monsanto and other biotech companies are profiteers bent on controlling the world's food supply. Canadian farmer Percy Schmeiser (right) came to see things more like activist Joe Parker (left) after becoming locked in a David-and-Goliath battle with Monsanto over his canola crop.

Monsanto accused Schmeiser of planting its Roundup Ready seed without being licensed to do so. Schmeiser responded that some of the seed he had saved from the previous season had been contaminated by Monsanto canola from neighbors' fields or spilled seed. In his ruling the judge said that Schmeiser infringed on Monsanto's patent by planting and selling seed he knew or should have known was Roundup Ready. According to the judge, it did not matter how the genetically engineered seeds got into Schmeiser's fields. The ruling distresses some farmers and activists, given the ease with which modified crops' genes can travel between fields. "Organic farmers can't even grow canola anymore," Schmeiser says. "It's all contaminated."

Contamination by engineered seeds is not restricted to canola. A recent study of 20 products labeled free of genetically modified (GM) ingredients found that 11 of them had traces of GM ingredients and 5 had significant amounts—and that's unintentional contamination. Such ingredients are routinely used in two-thirds of the processed food in the U.S. It may be too late to keep biotech and nonbiotech foods separate here, but Europe is trying. In 1998, incensed by regulatory failures that allowed mad cow disease to become a multibillion-dollar catastrophe, Europeans protested the "Frankenfoods" entering their markets. Major food retailers removed biotech foods from their shelves, and the European Union stopped approving new modified crops. Foods with more than one percent engineered content must be labeled.



SALVATION FOR SUBSISTENCE FARMERS?

For the Wathome family in eastern Kenya, biotechnology's risks pale next to its rewards. Before getting disease-free banana seedlings propagated from tissue culture, they barely had enough to eat. Now they sell a surplus. Genetically engineered sweet potatoes—more than a decade in the works—hold even more promise for millions of African farmers who depend on the disease-prone crop.

large companies that dominate the industry, critics also note, are not devoting significant resources to developing seed technology for subsistence farmers because the investment offers minimal returns. And by patenting key methods and materials, these companies are stifling the free exchange of seeds and techniques vital to public agricultural research programs, which are already under severe financial constraints. All of this bodes ill, say critics, for farmers in the developing world.

Prakash agrees that there's enough food in the world. "But redistribution is just not going to happen," he says. "The protest against biotech on political grounds is a straw man for a larger frustration with globalization, a fear of the power of large multinational corporations. People say that this technology is just earning profit for big companies. This is true to some extent, but the knowledge that companies have developed in the production of profitable crops can easily be transferred and applied to help developing nations."

Biotechnology is no panacea for world hunger, says Prakash, "but it's a vital tool in a toolbox, one that includes soil and water conservation, pest management, and other methods of sustainable agriculture, as well as new technologies."

The debate over the use of biotechnology in developing countries recently went from simmer to boil about rice, which is eaten by three billion people and grown on hundreds of millions of small farms.

"White rice," explains Dean DellaPenna, "is low in protein. It has very little iron, and virtually no vitamin A." However, in 1999 a team of scientists led by Ingo Potrykus, of the Swiss Federal Institute of Technology, and Peter Beyer, of the University of Freiburg, Germany, announced a new breakthrough: They had introduced into rice plants two daffodil genes

and one bacterial gene that enable the rice to produce in its grains beta-carotene, a building block of vitamin A. According to the World Health Organization, between 100 million and 140 million children in the world suffer from vitamin A deficiency, some 500,000 go blind every year because of that deficiency, and half of those children die within a year of losing their sight. "Golden rice," so named for the yellow color furnished by the beta-carotene, was hailed by some as a potential solution to the suffering and illness caused by vitamin A deficiency.

Skeptics consider golden rice little more than a public relations ploy by the biotechnology industry, which they say exaggerated its benefits. "Golden rice alone won't greatly diminish vitamin A deficiency," says Marion Nestle. "Beta-carotene, which is already widely available in fruit and vegetables, isn't converted to vitamin A when people are malnourished. Golden rice does not contain much beta-carotene, and whether it will improve vitamin A levels remains to be seen."

Potrykus and Beyer are now developing new versions of the rice that may be more effective in delivering beta-carotene for the body to convert to vitamin A. Their plan is to put the improved rices free of charge into the hands of poor farmers. According to Beyer, golden rice is still at least four years away from distribution. It could take much longer if opposing groups delay plans for field trials and safety studies.

Q: What next?

A: Proceed with caution.

Whether biotech foods will deliver on their promise of eliminating world hunger and bettering the lives of all remains to be seen. Their potential is enormous, yet they carry risks—and we may pay for accidents or errors in judgment in ways we cannot yet imagine. But the biggest mistake of all would be to blindly reject *or* endorse this new technology. If we analyze carefully how, where, and why we introduce genetically altered products, and if we test them thoroughly and judge them wisely, we can weigh their risks against their benefits to those who need them most. □

MORE ON

Sound off on the benefits and risks of biotech food in our forum and check out an Online Extra at nationalgeographic.com/ngm/0205.



U N I C O

A close-up photograph of a moth's wing, showing intricate patterns and iridescent colors. The wing is divided into several sections by dark veins. The colors range from deep purple and blue to vibrant green and yellow. The texture of the wing appears fine and detailed.

METALLIC
WINGS.
GOLDEN
ANTENNAE.
YES, IT'S
A MOTH.



M M O N
VISION

BUT MOTHS ARE SWEATER-MUNCHING, LAMP-



SWARMING, DUST-COLORED PESTS, RIGHT?

NOT TO ARTIST JOSEPH SCHEER. *"They're the wicked stepsisters of the butterflies,"* he says—part of a nighttime world most of us rarely see. He calls comically patterned *Arctia caja* (below, wingspan 2 to 2.8 inches) *"a moth with presence."* Co-founder of Alfred University's Institute for Electronic Arts, Scheer intends to create a digital portrait of at least one moth from every species that lives in—or even passes through—western New York's Allegany County. He met *Diachrysia balluca* (preceding pages, wingspan 1.6 to 2 inches) in 1999, his first collecting season. *"Just one,"* he recalls. *"But it was in perfect condition. Gleaming green wings, furry tufts down the body, gold wire antennae. Really exquisite."* Three years and a thousand species later Scheer remains cheerfully moth-obsessed.



IMAGES BY
JOSEPH SCHEER

TEXT BY
LYNNE WARREN

NATIONAL  WRITER



Seemingly invented out of conchids, flower petals, and stained glass, two dozen species of moths swarm a 15-foot-high wall in Scheer's studio. Exhibited from Europe to the Far East, the prints inspire one consistent reaction: disbelief. "People insist, 'No, that can't be a moth,'" says Scheer. A Swiss viewer credited the insects' glamorous variety to their exotic American origin: "We don't have such nice moths in our country," he declared. (Not so. Every country has moths that can dazzle.)



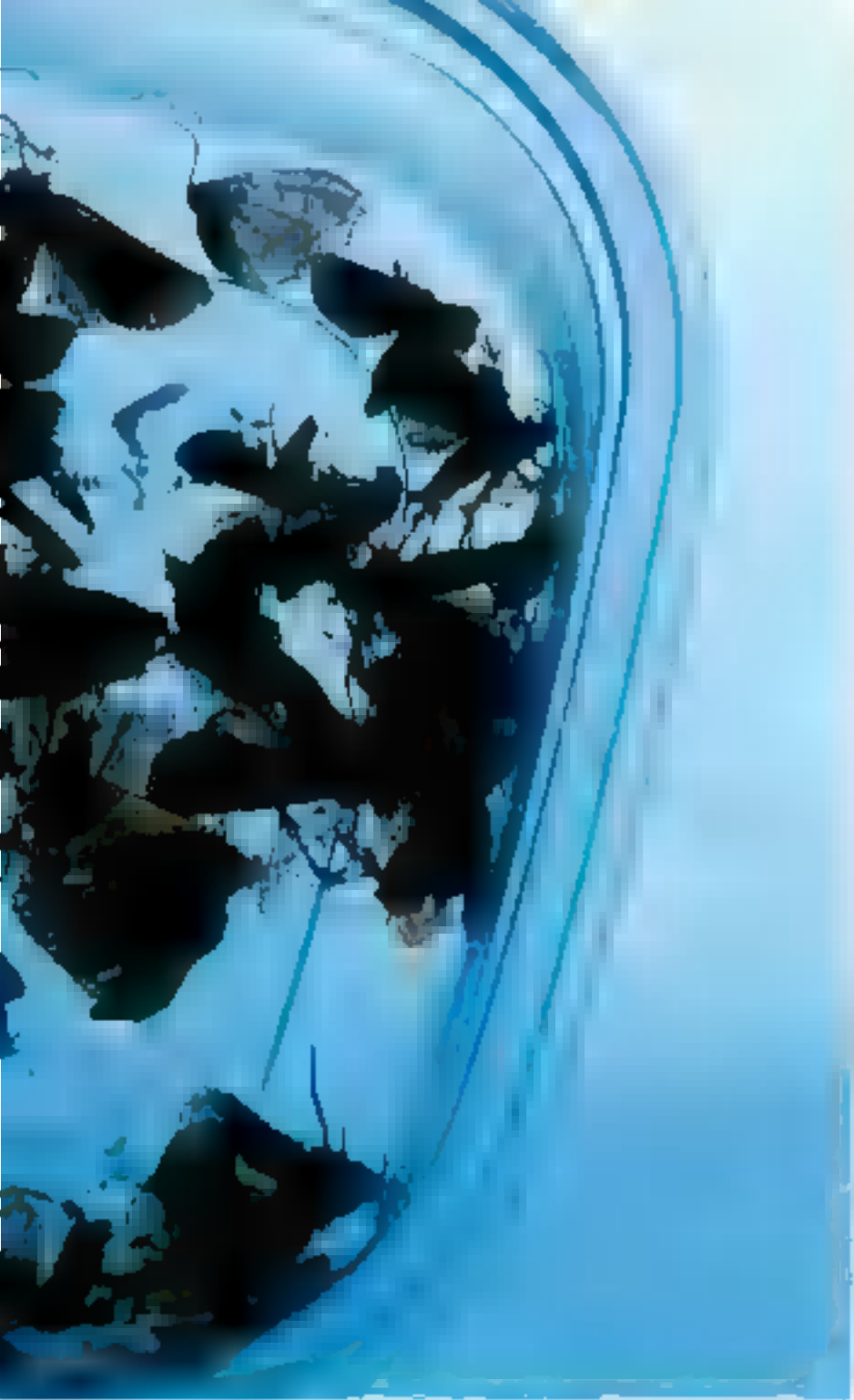


The moth hunt began with Joseph Scheer using his Alfred University office as a lure, leaving lights on and windows open at day's end, collecting whatever had wandered in overnight when he returned the next morning. Plenty of moths showed up, but janitors howled about the buggy mess. So Scheer moved the hunt to his colleague Mark Klingensmith's yard. "Mark's a gardener with lots of stuff growing on his property," Scheer says. "Moths like it." The two set up lights glowing over a five-gallon bucket and shining on a white sheet (above left, with Klingensmith standing). Then they watched, astounded, as moths came looping, fluttering, zooming in. "We got a different species every night that first season," Scheer says. "The patterns and colors were overwhelming."

A technical specialist at the university's Institute for Electronic Arts, Klingensmith coaxed a scanner designed for film and transparencies into capturing pictures of three-dimensional moths. The scanner records so much information—67 million data points per square inch—that a single specimen may take 20 minutes to scan. The data files generated are huge: Two small moths fill an entire compact disc.

With resolution that high, scans can be enlarged 2,700 percent and still be perfectly clear. Moths that in life rest comfortably on a fingertip splash across 34-by-46-inch art papers. You'd need a microscope to see the tiny scales on body and wing as clearly as they're revealed in Scheer's prints. At every step from scanner to monitor to printer, the artist keeps the actual specimen in front of him, constantly comparing his digital representations to nature's original. "Every moth requires hours of work," he says. "Color correcting the scan, adjusting the printer so the final image truly matches the moth. It has to be perfect."

Scheer and Klingensmith improve their self-taught bug-handling skills from season to season. The ultras-small moths called microlepidoptera



IRA BLOCK (ABOVE, ALL)

present special challenges. “One twitch of a finger and there goes a wing,” Scheer admits. “I try to drink less coffee when I’m working on micros.” Even steady-handed, green-tea-drinking Klingensmith barely breathes as he arranges moths of various sizes on the scanner, holding them steady against the glass using tiny weights lowered onto the wings with forceps (above right).

Collecting starts on the first warm evening in April and stretches through the last frost-free dusk in November. Each night’s bounty goes into plastic sandwich boxes (above center), with a little alcohol to keep the specimens moist and flexible until they can be pinned in preparation for scanning. “It’s the first thing Mark and I talk about in the morning,” Scheer says. “What did we get in the bucket last night?” Moth-filled containers proliferate in refrigerators at home and at work. Stacked on every available surface, foam blocks bristle with pins anchoring moth wings. Moth CDs line shelves and fill cardboard boxes on the floor.

To transform this moth mass into a meaningful collection, Scheer enlisted the aid of Marc Epstein, a collections manager for the Smithsonian Institution’s National Museum of Natural History. Epstein has spent much of his life studying Lepidoptera, the insect order that includes butterflies and moths, but he was “stunned by the amount of detail” captured in Scheer’s images. “Joseph has created his own art form, his own way of visualizing moths and sharing what he sees with other people,” the scientist says.

With Epstein’s guidance Scheer has cataloged and identified his subjects. What began as an art project has also become a scientifically valuable record of moths in Allegany County: some 15,000 specimens, representing more than a thousand species. “Not from Alaska or the Amazon,” Mark Klingensmith says. “All from one backyard. It’s an ordinary yard—but look at the amazing diversity we’ve found there.”

The luna (Actias luna) is a big, strong moth, with wings three to four inches across. But its flying life is short—just a week or two, spent dodging hungry birds and bats. Its beauty can be even more fleeting. “As soon as we collect a luna the color starts to fade,” Scheer says. “We have to get it on the scanner almost instantly to capture that luminous green.”



MORE ON OUR WEBSITE
Peruse a gallery of Joseph Scheer’s moth portraits and learn about websites devoted to moths at nationalgeographic.com/ngm/0205.
AOL Keyword: NatGeoMag





*Blue eyes, coyly curled antennae, and a hot pink feather-boa wrap suggest design influences from Barbie doll outfits and pop diva music videos. At life-size the wings of this small show-off—*Hypoprepia miniata*, the scarlet-winged lichen moth—span just one inch. Creating portraits so much larger than life gives his flamboyantly costumed night fliers “a kind of hyper-reality,” Scheer says. “Digital tools let you see things you’d never see just looking with your eyes.”*



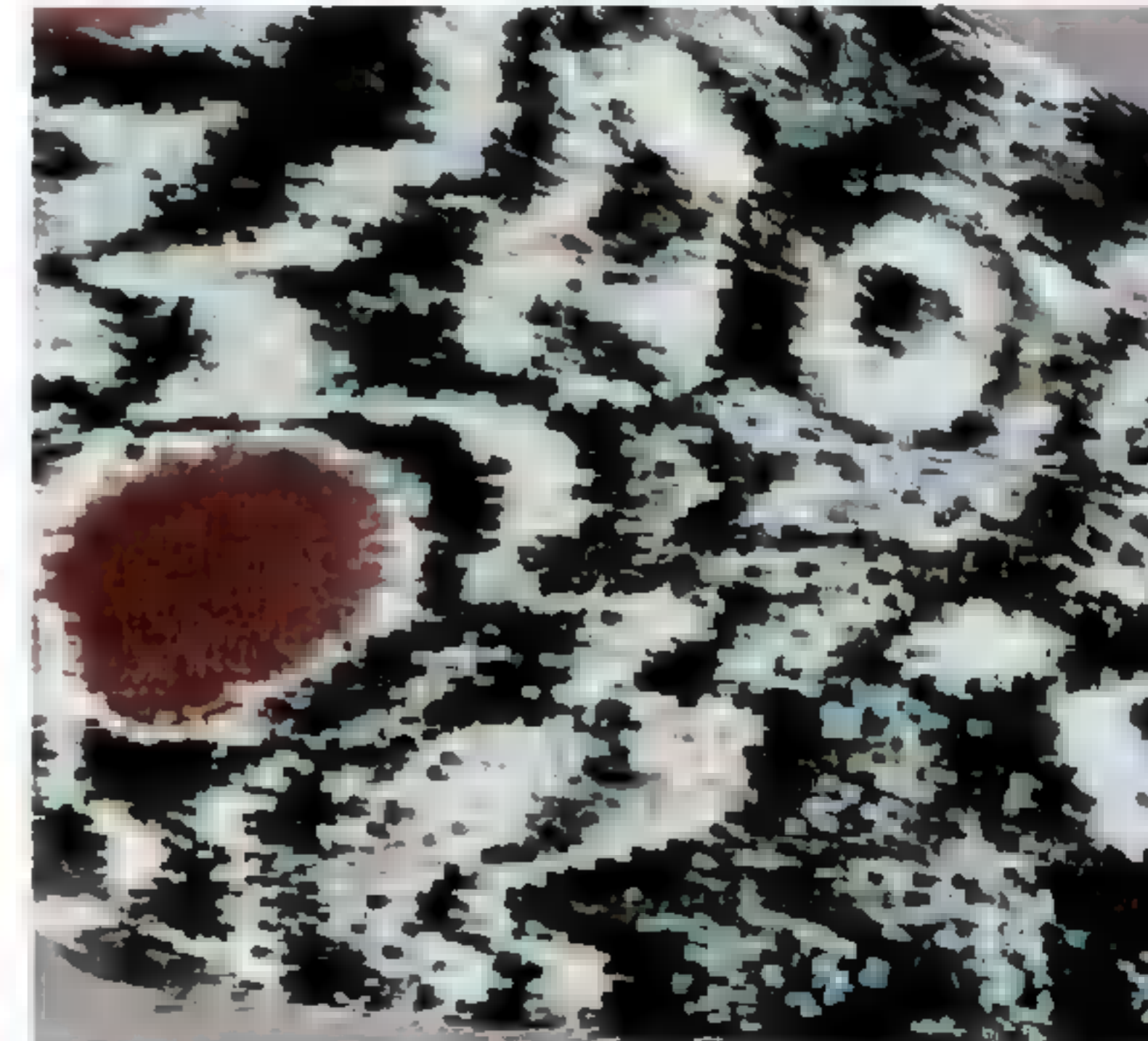
THEY'RE GAUDY, PLUSH, ELEGANT, ODD.



Peoria approximella, wingspan 0.6 to 0.8 inches



Phylodesma americana, wingspan 1.1 to 2 inches



Harrisimemna trisignata, wingspan 1.2 to 1.4 inches



Campaea perlata, wingspan 1.1 to 2 inches

THE CLOSER YOU LOOK, THE MORE YOU SEE.



Paonias myops, wingspan 1.5 to 3 inches



Automeris io, wingspan 2 to 3.1 inches



Synanthedon exitiosa, wingspan 0.6 to 1.4 inches



Zeuzera pyrina, wingspan 1.8 to 2.8 inches



Antheraea polyphemus, wingspan 4 to 6 inches



CATFISH HUNTERS

In the name of science, they splash around in tropical rivers. They stick their hands into underwater logs. They have no idea what's in there. And they swear it's fun.

*By BEN MONTAGONE
Photographs by RANDY OLSON*



How are the fish faring in Guyana's threatened rivers? To find out, scientists went down for the count—tallying all the fish they could find, including this suckermouth armored catfish.

JUST UPSTREAM of the ramshackle jungle outpost of Tumatumari, two local men and a boy stood knee-deep in the brown waters of the Potaro River and stared at an odd gathering of foreigners. Against a backdrop of Guyana's dense rain forest and the concrete remains of a defunct hydroelectric dam, three young biologists whooped and wallowed in the warm river, rolling boulders, stomping clumps of river vegetation, and dragging nets through the stream. They were in search of fish, the rarer—and uglier—the better.

Jonathan Armbruster, a jungle river veteran, slithered on his ample belly along the stream bottom, a swim mask on his face. Armbruster, 29, has a special fondness for "hogging," a practice in which the collector worms his fingers into submerged logs to pry loose reclusive fish.

"Smokin' hassar!" shouted Armbruster using the local name for a suckermouth armored catfish. He held aloft a four-inch brown creature with a sail-like dorsal fin, a homely specimen only an ichthyologist could love.

"You gonna' cook dem?" the 12-year-old villager, Leon Lewis, asked as Armbruster tossed

the catfish into a white plastic bucket. It was a logical question, but logic did not always apply to this expedition, which was led by a United States fish biologist who, of all things, did not know how to swim.

The two Guyanese, both gold miners, joined in the fun on the Potaro, holding the seines as the biologists tromped about the river. It poured rain, but no one cared, for the river and the rain were the only relief from the enervating heat.

I asked one of the miners, Andrew George, what he thought of these young scientists. George—a slender, tautly muscled 30-year-old wearing only a pair of blue-jeaned shorts—pondered the question a few seconds, then replied, "They catchin' fish. It's a lot of fun catchin' fish."

Fun indeed! For love of the lowly catfish, these scientists slogged through the jungle in hundred-degree heat, impaled themselves on the three-inch thorns of spiky palm trees, and endured the bites of myriad insects. In the end they brought back to the U.S. a motley cast of creatures, including fish with teeth so long they poked through their upper jaws and protruded from their skulls.

"There's so much cataloging still to do here," said Armbruster, an assistant professor of ichthyology at Auburn University in Alabama. "In North America people have collected just about everything. Down here the collecting is so sporadic you hardly have any areas that are well studied."

SOCIETY GRANT

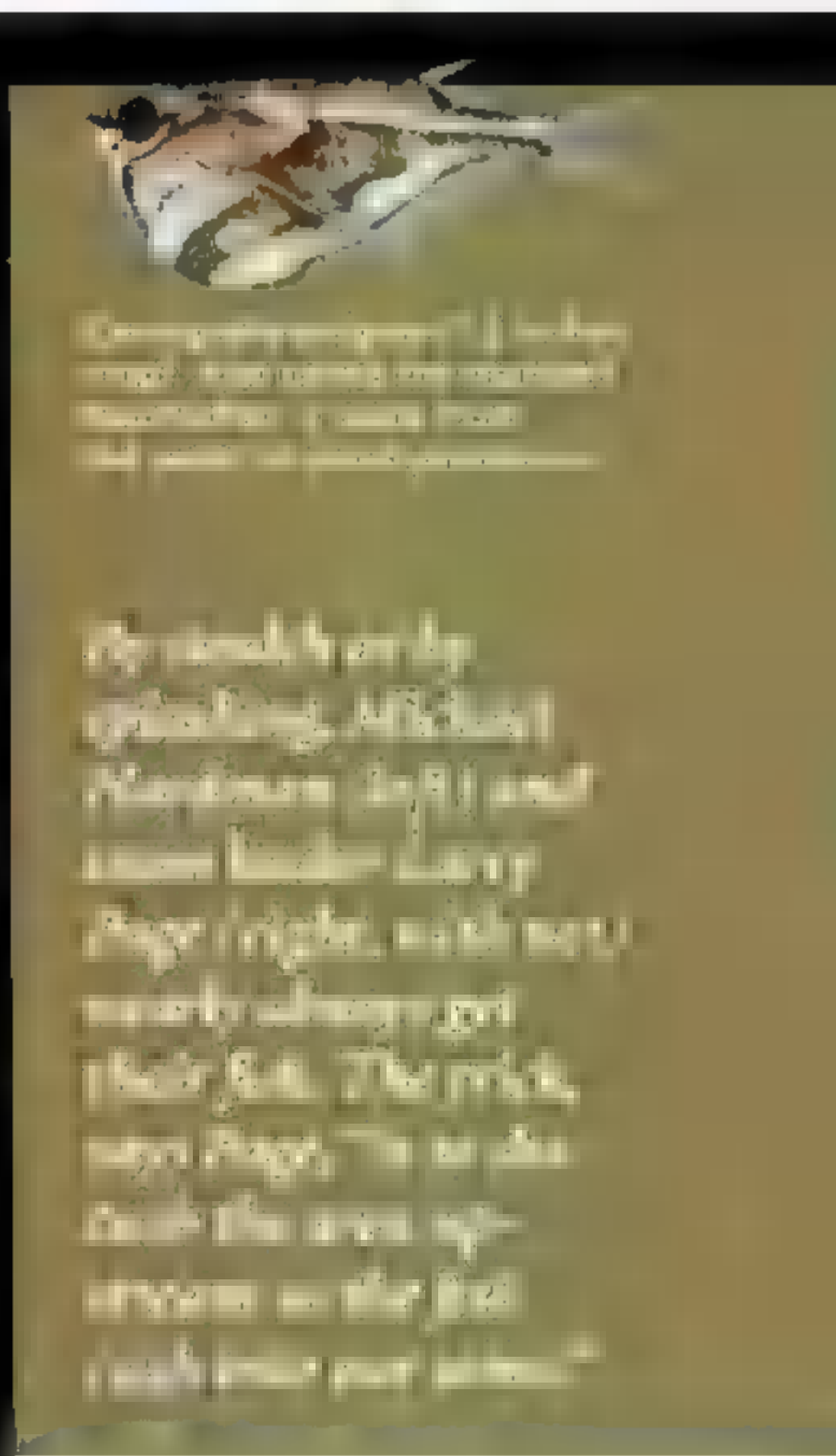
This Research Committee project is supported by your Society membership.





The 741-foot Kanabec Falls—four times taller than Niagara Falls—slices through Geyser's wilderness, its frothy surging into and clear-cut. Scientists saw roughly the same number of fish as did Carl Eigenmann, who surveyed these waters in 1908.

CASTING ME A WRY LOOK, OUR LEADER SAID, "I SHOULD HAVE STAYED AT THE MUSEUM COUNTING FISH SCALES."



The expedition was the brainchild of Lawrence M. Page, 54, a professor at the University of Illinois and principal scientist at the Center for Biodiversity at the Illinois Natural History Survey. A non-swimming veteran of a dozen ichthyological expeditions to Central and South America, Page hatched the idea of retracing the route of Carl H. Eigenmann, an American scientist who, in 1908, traveled up the Essequibo and Potaro Rivers in what was then the colony of British Guiana, collecting 336 species of fish. Although Guyana (an Idaho-size nation with a population of 800,000) has one of the largest untouched expanses of rain forest in the world, recent logging and gold mining have begun to sully its waterways. The goal of Page's expedition, financed by the National Geographic Society, was to determine what had happened to the fish in these two rivers and to get a sense of how polluted they had become in the years since Eigenmann's visit.

"You guys better plan for the worst," a veteran bush pilot with the delightful name of Malcolm Chan-A-Sue told us on the eve of our departure, "because that jungle ain't changed much since that man Eigenmann's been there."

The expedition began in earnest at Rockstone, a tiny outpost hacked out of the rain forest on the Essequibo River. As we approached the landing on a jungle-lined dirt road, scattering clouds of yellow and chartreuse butterflies, we saw a group of more than a dozen women and children, dressed in long skirts and colorful blouses. They piled into an old wooden boat and, eight dugout canoes in tow, headed upstream, where they would spend two days catching piranha, catfish, arawana, and angelfish for the international aquarium trade.

The next morning our team of eight (six Americans, an Englishman, and a Guyanese) got into a small motorboat and explored a canal off the Essequibo. Thick white clouds drifted across the sky, occasionally blocking a sun so hot that the mere act of sitting induced a shirt-soaking sweat. Red and green kingfishers shot in front of our wooden boat. Iridescent hummingbirds hovered over the cove. As we drifted into a fetid lagoon clogged with dead trees, a snake skittered across the water. Armbruster pronounced conditions perfect. He was the first over the side.



The biologists engaged in a hogging frenzy, probing half-submerged logs. Michael Hardman, a 24-year-old British Ph.D. student at the University of Illinois, was the first casualty. “Ohhhhh, @#@#!” he screamed. Wading through the hot, chest-deep water, he had placed his hand on a submerged spiny palm, and a half dozen of its black needles had broken off in his hand. But his suffering was not in vain: A few minutes later the team hoisted a ten-foot hollow log containing so many fish it rattled like a maraca.

Armbruster was into the log up to his shoulder when he grabbed a fish that took a liking to his hand. “What is that?” he hollered. “My fingers are meat.”

In rapid succession the scientists extracted a tetra, a rare nine-inch catfish, and a spiny catfish oozing a milky poison from its skin. The U.S. and Canada contain approximately 40 species of catfish. South America has about 1,200 different catfish species, roughly half the world’s total. In Guyana alone there may be as many as 300.

It was late October, ostensibly the dry season. That afternoon it poured, flooding our tents.

“Dry season,” we were discovering, was a cruel misnomer. As Jason Knouft, also a Ph.D. candidate at Illinois, put it, “There is no dry here, just different degrees of damp.”

We headed up the mile-wide Essequibo the next morning in a battered light-blue wooden boat about 20 feet long, shaded by a green plastic tarp stretched across a jury-rigged bamboo frame. The Essequibo, Guyana’s largest river, flows out of the Acarai Mountains on the Brazilian border, then streams north 630 miles to the Atlantic Ocean. Though biologists are busy studying the loss of plants and animals in the rain forests of the world, including Guyana, little is known about the tropical fish that inhabit rivers like the Essequibo.

“You have to know what’s there so that when people want to draw up a conservation plan, there’s some data for them,” said Armbruster, who has kept aquariums of exotic fish since he was a boy. “We want to preserve these fish, but you can’t do that blindly.”

That night, as we pitched our tents on a sandbar, towering thunderclouds formed to

“THIS WHOLE RIVER USED TO BE CLEAR, AND LOOK AT IT NOW,” SAID THE VILLAGE HEADMAN.

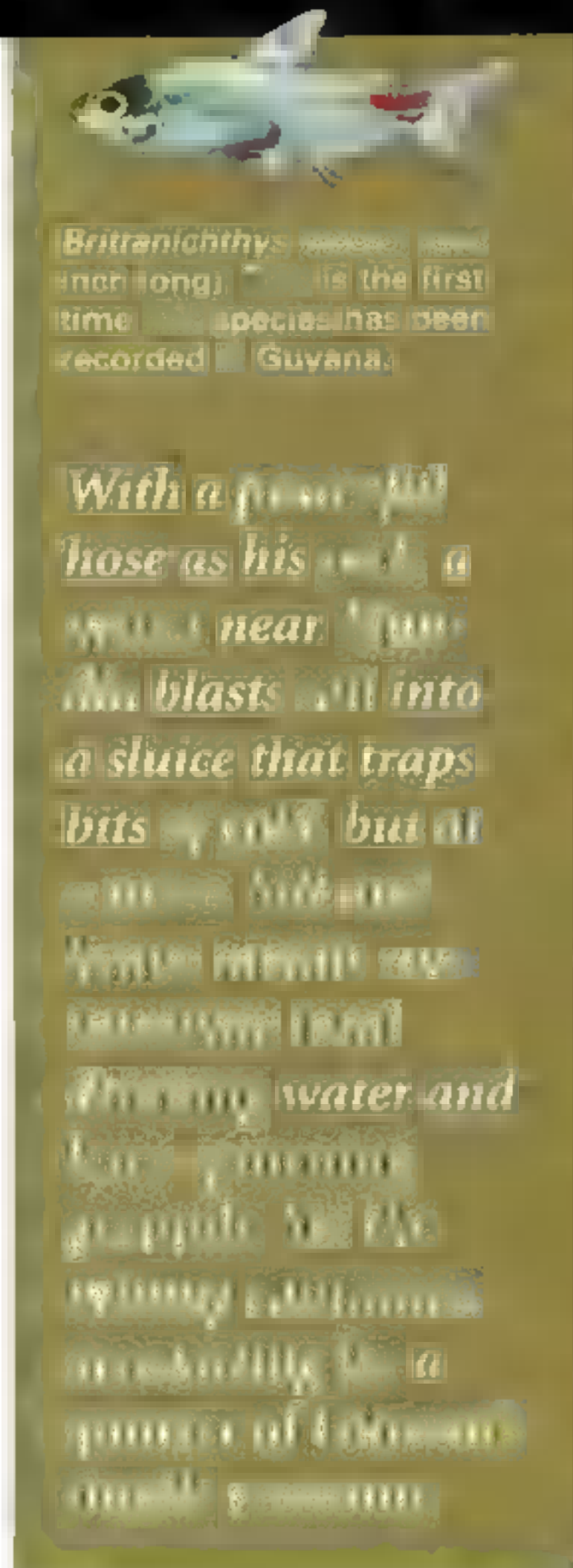
the east. Just after dark, lightning arced over the jungle. The storm blew past, and the team grabbed nets and flashlights and seined a nearby lagoon under a black sky lit with stars. The haul was bountiful: hundreds of *Corydoras* catfish; a handful of large catfish; long, slender, translucent knifefish; scores of silver tetras that shimmered in the nets.

In the morning Mark Sabaj, a 29-year-old Ph.D. student from Illinois, made a mistake none of us would make again: He put a foot in his tennis shoe without first shaking out the bugs, and was stung. Yelping, he flung off his shoe, and out came a one-and-a-half-inch-long bullet ant. Sabaj might as well have been shot; for the next two hours, as we motored upstream, he was in agony.

So far we had seen relatively unmolested stretches of the Essequibo, but that soon changed as we motored into the heart of Guyana's gold-mining territory. First we passed Omai, a town of trailers and prefab buildings that is the site of the largest gold mine in Guyana. There, in 1995, a huge tailings impoundment—contaminated with the sodium cyanide used to separate gold from other minerals—collapsed, spilling 800 million gallons of wastewater into Omai Creek and the Essequibo. The spill led to large fish kills on the river.

Above Omai we left the Essequibo and turned into the Potaro River, along which Carl Eigenmann had collected many specimens. The Potaro plunges down from Ayanganna Mountain near the Brazilian border and drops more than 6,000 feet in its 140-mile course to the Essequibo. We were disappointed to see that the lower Potaro—once renowned for its clarity—was as murky as the Essequibo. Soon we found out why: Gold dredges, operated by small owners, were stationed in the river, using great vacuum-like devices to suck up sand and silt from the bottom. The sand is then sifted for gold and dumped back into the river, a process that buries fish spawning grounds and creates chains of man-made sandbars.

Faced with a series of long portages, we



left behind our old boat and briefly traveled overland, passing through malaria-infested gold-mining camps. We returned to the Potaro a day later, hiring new boats at Pamala, little more than a one-room trading post sitting atop a jungle bluff. From there we could see the beginning of the remote Guyanese highlands, which would eventually lead us to Kaieteur Falls, four times higher than Niagara.

With renewed zeal, the ichthyologists picked up their seines. But at Amatuk Falls, where water thundered over a 30-foot drop, the results were disappointing. Eigenmann had collected 87 specimens of a catfish known as *Lithoxus lithoides*, but our team found none. The reason, the scientists speculated, was an accumulation of mining by-products, including mercury, that harm bottom-feeding fish.

We had hoped the heat would ease as we ascended into the highlands, but there was little relief. Waldyke Prince, the Guyanese biologist, kept relatively cool by sleeping in a hammock



with a mosquito net. The rest of us slept in tents, and soon we were sorry we hadn't gone native. With little air stirring, I usually spent the first hour or two sweating prodigiously. The next hour I spent scratching the chigger and sand flea bites that had erupted like measles spots on my legs and midsection. "My time for not sweating," photographer Randy Olson announced after one particularly miserable night, "is between 3:15 and 4:15."

After a punishing portage around Amatuk, Page and I sat in a boat and watched with weary disbelief as our skipper struggled—unsuccessfully—to start our engine. We were 200 yards above the falls, drifting steadily toward the precipice. I was beginning to contemplate a swim for shore, but Page had no such option. Casting me a wry look, our leader said, "I should have stayed at the museum counting fish scales." A hundred yards above the falls, the boat captain coaxed the engine to life.

Chugging upriver through the Potaro Gorge,

where steep, tree-covered hills closed in on us from both sides, we reached the foot of Kaieteur Falls. Not far from our camp, we came upon a grove of cashew trees bearing yellow-orange, pear-shaped fruit, atop which was the husk of the cashew nut. The group hadn't eaten fresh fruit in a couple of weeks, and we devoured the astringent, yet sweet cashew pulp. Sabaj, the bullet ant victim, was the fittest scientist on the expedition, but he was also the unluckiest. He experienced a massive allergic reaction to the fruit and soon was on his back in his tent, his face, hands, and feet grotesquely swollen.

That afternoon, a dozen Amerindians—short, sinewy men wearing a ragtag assortment of pants and cutoffs—moved silently into our camp. They were from the village of Chenapau, upriver from the falls. Carrying bows and arrows, spears, dried cassava bread, and woven baskets for backpacks, the men would help us carry our gear to the top of the falls.

*THEY PASSED AROUND THE LITTLE FISH.
WHEN IT RETURNED TO ARMBRUSTER, HE
KISSED ITS PUCKERED MOUTH.*



After visiting 272 species, including a special European fish, Eigenmann was struck by the beauty of the little fish.

After visiting 272 species, including a special European fish, Eigenmann was struck by the beauty of the little fish. He was so awestruck by Kaieteur that, despite a high fever from malaria, he clambered onto a ledge and set up his tripod camera. "I confess to feeling distinctly dizzy when I placed my head under the focusing cloth, knowing that if something should happen I and the camera would land on the rocks . . . below," he wrote. "Not that I could find a finer place to die, but I was reluctant to start to "kingdom come" on such a heavy down grade!"

"This whole river used to be clear, and look at it now," said Anthony Melville, the village headman, who had a curly head of black hair, a sharply chiseled nose, and a wispy mustache. "Nobody cares a damn as long as they have money in their pockets."

The next morning we set off for the top of Kaieteur Falls, following a narrow trail that wound upward through a dim jungle. Our steep path crossed small streams and long lines of leafcutter ants marching to yard-high nest mounds in the decadent tropical understory. Stopping for a break, we wrung cupfuls of water out of our shirts. After two hours we emerged from the forest, crossed a swath of savanna on the plateau, and stood at the top of one of the world's most spectacular waterfalls.

At Kaieteur the Potaro cascades 741 feet in one drop over the sandstone ledge that separates Guyana's highlands from its sea-level rain forests. The wide light-brown sheet thunders down with such force that it kicks up clouds of mist that drift upward above the falls. I stood atop the falls and surveyed the jungle, which stretched unbroken to the horizon. (Thanks to low population density and a

previous socialist government that discouraged foreign investment, Guyana is still 80 percent forested.)

The view had not changed much since Eigenmann's day. As he stood there in 1908, the scientist was so awestruck by Kaieteur that, despite a high fever from malaria, he clambered onto a ledge and set up his tripod camera.

"I confess to feeling distinctly dizzy when I placed my head under the focusing cloth, knowing that if something should happen I and the camera would land on the rocks . . . below," he wrote. "Not that I could find a finer place to die, but I was reluctant to start to "kingdom come" on such a heavy down grade!"

Our final destination was Chenapau, a village of thatched-roof lean-tos on a bluff above the dark waters of the Potaro. Its 500 inhabitants survive by subsistence farming, hunting, fishing, and mining. When we arrived, women were pounding cassava root to prepare an airy dried bread.

The next morning our team disembarked below a series of gentle cataracts, not far from where Eigenmann collected two rare species of



catfish—an armored specimen, *Corymbophanes andersoni*, and another flat, two-inch catfish with a gaping mouth, *Lithogenes villosus*. The scientists began rolling stones and beating river grasses, but nearly two hours of work yielded nothing.

Moving further upriver, we stopped at a wide riffle. Two of the fish biologists stretched a net across a 25-foot channel in the bedrock. Armbruster and Hardman waded in, furiously kicking the bottom. The men dragged the net to a rock and, spreading open the seine, sifted eagerly through pebbles, blades of grass, and a few common fish. It looked like another dry run. Then, in a voice that carried far down the Potaro, Armbruster shouted, “YES! *Corymbophanes andersoni*!”

The creature in question was a dark gray, two-and-a-half-inch, suckermouth catfish with light spots and tiny eyes. To me it looked like many of the oddball species of catfish we’d seen for weeks, but to Armbruster and Page it was submerged treasure, the first specimen found since Eigenmann. The scientists passed around the little fish. When it returned to Armbruster, he kissed its puckered mouth.

In three weeks of collecting, the catfish hunters netted 272 species of fishes. Overall, despite localized mining pollution, Page said that fish populations in the Potaro and Essequibo have held up fairly well since Eigenmann’s expedition.

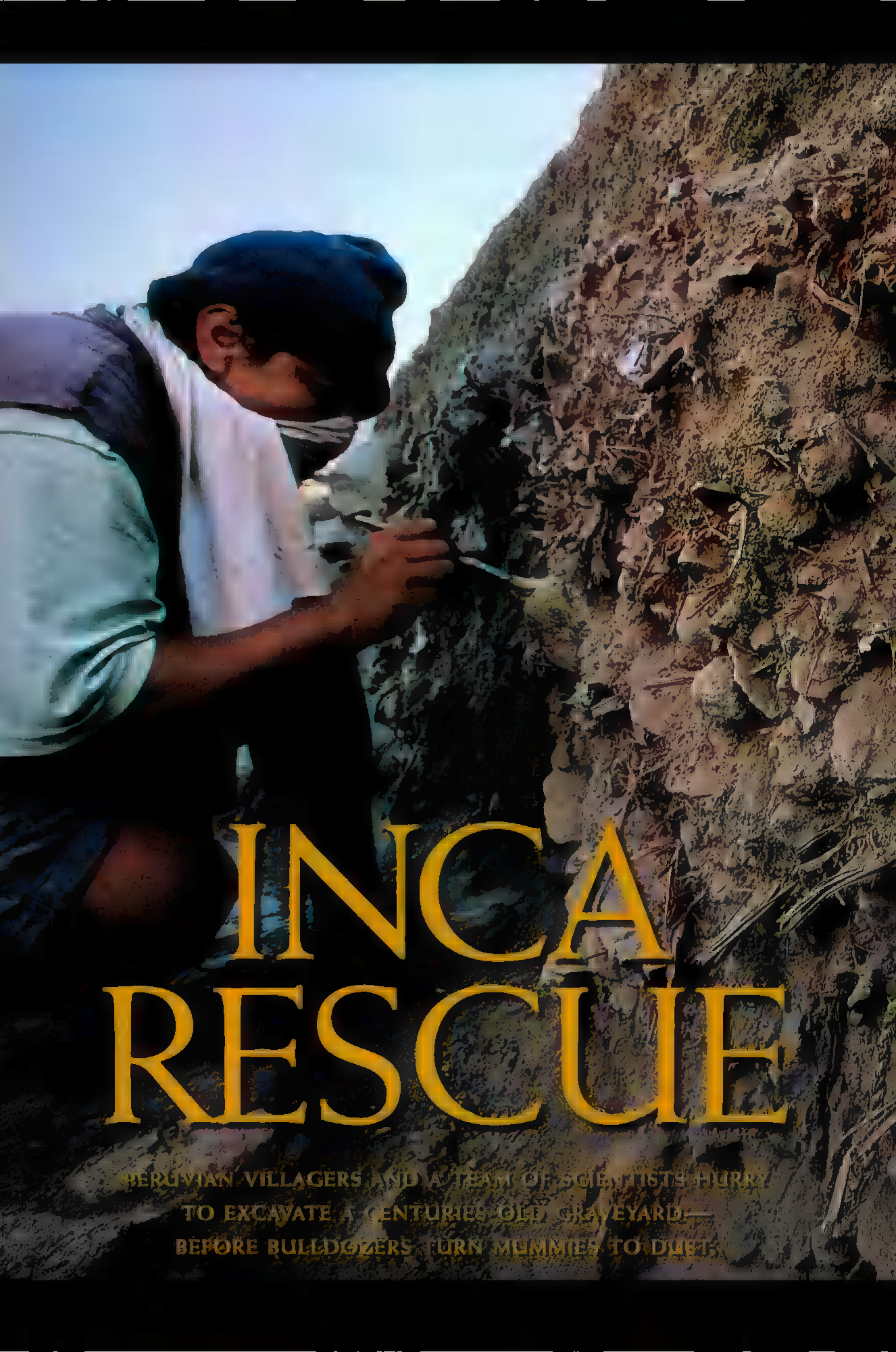
He worries, however, that a predicted increase in mining and logging could be bad news for the rivers, and the fish. In any case, Page said that his group, which also discovered at least six new species, accomplished its goal: to provide, along with Eigenmann, a benchmark “against which the impacts of future development in Guyana can be measured.”

That afternoon the group continued collecting. With each new rarity plucked from the nets, the scientists hollered in triumph. Standing in the Potaro, Anthony Melville chuckled as he contemplated the passion that gripped these foreigners.

“I never saw people interested in fish so small,” he said. “This size fish would pass right through your teeth.” □

MORE ON OUR WEBSITE

See more images of the catfish hunters and read about life on assignment at nationalgeographic.com/ngm/0205.
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INCA RESCUE

PERUVIAN VILLAGERS AND A TEAM OF SCIENTISTS HURRY
TO EXCAVATE A CENTURIES-OLD GRAVEYARD—
BEFORE BULLDOZERS TURN MUMMIES TO DUST.



By GUILLERMO A. COCK
Photographs by IRA BLOCK

In a sprawling shantytown called Tupac Amaru (far right) on the outskirts of Lima, children play in the dust of ages. Beneath their feet, preserved by the bone-dry soil, lies one of the largest Inca cemeteries yet found in Peru. This pre-Hispanic site, known to archaeologists as Puruchuco-Huaquerones, dates from the Late Horizon (1438 to 1532). Though it has been designated a national monument, my team of scientists has had to race against bulldozers to pull the past out from under the burgeoning present. Beneath the schoolyard alone (bottom right), one of 15 areas examined in three years, we've salvaged more than 120 mummy bundles (layers of cloth encasing a body and personal effects) typical of pre-Inca and Inca burials.

The story of how Tupac Amaru came to be is a common one in Peru. In 1989 some 340 families fleeing guerrilla activity in the highlands settled on this property, misled by land traffickers to believe they would soon be given title. Meanwhile, six feet under and defenseless against the sudden influx of sewage and water, the mummies were decomposing. Some squatters dug them up and burned them, hoping to avoid a scientific excavation that would delay town development.

Though much damage was done in subsequent years, the Peruvian Institute of Culture



(INC) finally did request an archaeological evaluation of the area. I arrived from Lima in 1999, tools and team in tow. Not wanting to be relocated by the government, the townspeople—then more than 1,240 families—agreed to stop leveling the land and even scraped together money to help fund our work. They hoped it would be a long-term investment, encouraging the government to give them what the traffickers couldn't: clear land titles and basic utilities.

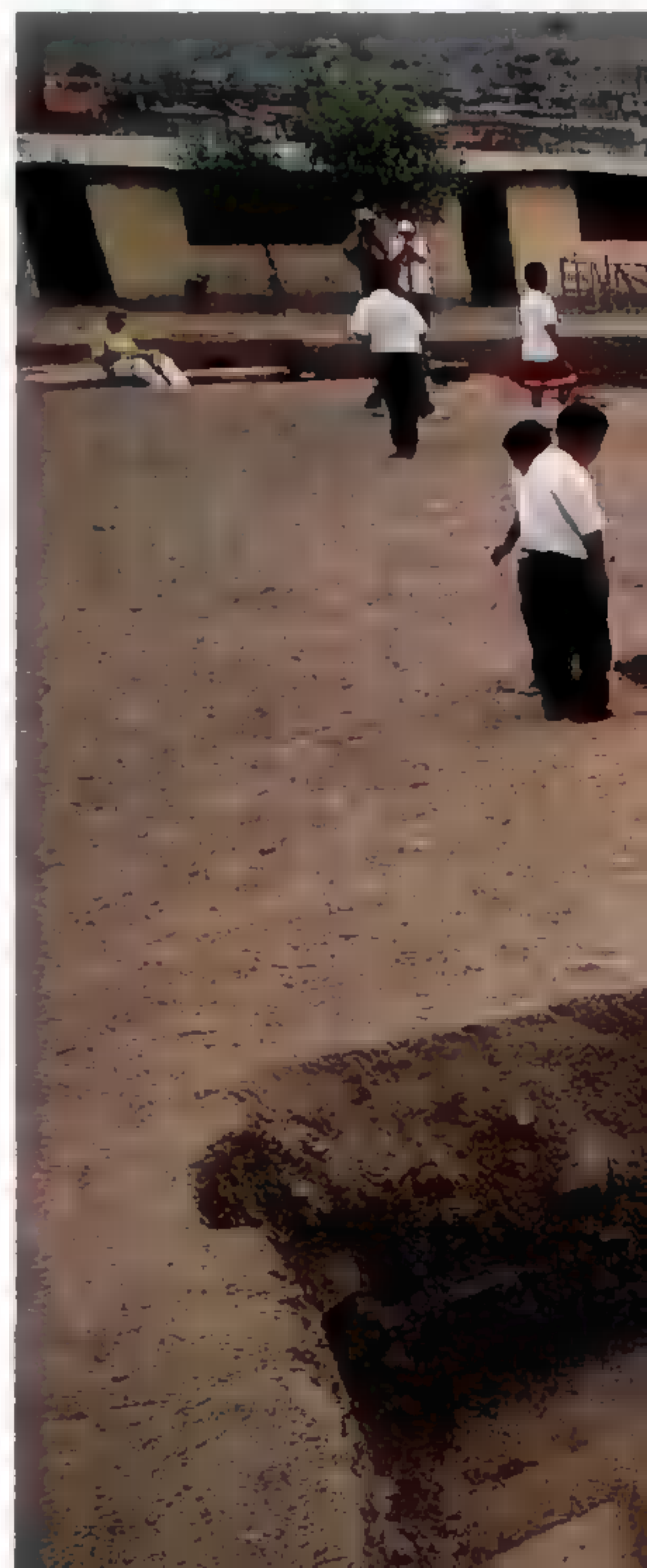
At first the residents assumed we would loot the tombs or dig briefly and halfheartedly, pocketing leftover funds. But we hired locals to help excavate, soon earning their trust. In addition, the INC visited the site weekly. In three field seasons we've removed, examined, and photographed more than 2,200 individuals of all ages and ranks buried within 75 years of one another. At 20 acres this is the second largest cemetery ever excavated in Peru (after Ancón) and the largest from a single time period. A local museum will ultimately display these cultural treasures.

As we continue to delve

into the past, life in Tupac Amaru flourishes. Children frolic on sacred ground, running along the rims of our excavations, peering into the tomb of a dead man who has claimed their soccer ball. Some believe the spirits of the deceased have caused a spate of illnesses here, including my own lingering cough. Indeed, inhaling dust of the dead can lead to bacterial infection. Yet many say they are not fearful but elated by their privileged view of those who walked this land before them.

ON OUR WEBSITE

Find out why locals think these mummies have cursed the living. Explore this and more at nationalgeographic.com/inca.
AOL Keyword: NatGeoMag



SOCIETY GRANT

This Research Committee project is supported by your Society membership.





Secrets Under the Schoolyard

A village priest hid a massive, undisturbed mummy bundle for removal from the excavated schoolyard. It took four men and a sturdy plank to raise the 180-pound bundle from its grave without breaking the brittle bones inside. We call such mummy bundles *falsas* after the false heads (textiles stuffed with cotton) placed on top. Excavated nearby, a sign of high status, still clinging to the wall of a mummy tomb nearby (right). The copper star (above), unearthed south of the tomb, once embellished a wooden child made of cane and reeds. The dry climate helped maintain these treasures, but so did the people who buried them—by sealing the tombs with sand, rubble, and ceramic sherds.



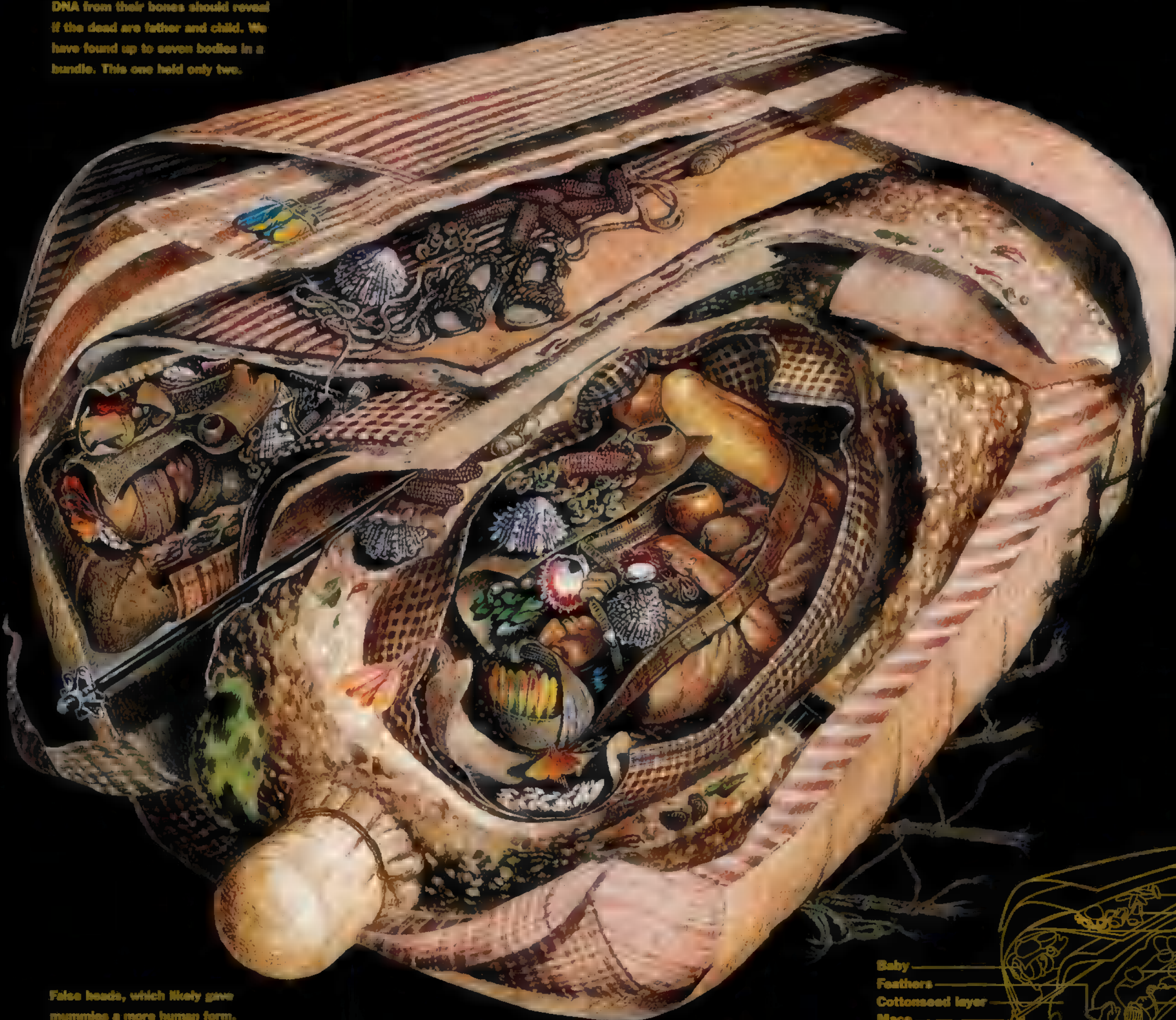


A Funerary Bundle Unfurled

We nicknamed one truly unique mummy the "Cotton Mummy" because he was bundled in over 500 pounds of heavy cotton. Typically, the Inca wrapped nobles in fine llama or alpaca wool. In this case, members of the team were still in the process of excavating the site with their fingers (below), and many ancient artifacts were left buried within. A highly prized, related, shared life bale; the mummy was found in a small tin in the cotton. The extensive packing and the many items buried with the mummy suggest an elite status.



DNA from their bones should reveal if the dead are father and child. We have found up to seven bodies in a bundle. This one held only two.



Their souls were thought to keep in touch with the living, so the Inca dead were well tended. The Cotton King was buried with everyday items—food, pottery, animal skins, and corn to make *chicha*, a fermented drink. Other objects signified his high place in society: Exotic bird feathers adorned his headband, which doubled as a slingshot; his mace, or club, signified a powerful warrior; and his sandals were a type worn by the upper class. Most revealing of his wealth were offerings of *Spondylus* oyster shells imported from warm waters off the coast of Ecuador.

The baby who shared his bundle lay with its legs straight out, but the man's pose, like the cotton stuffing, puzzled us: Rather than tucked in the fetal position typical of adults, his knees were bent as if kneeling, his toes pointed like a dancer's (pages 88-9). Is there meaning in this eccentricity? We aren't yet sure.

PHOTOGRAPHY
 See each layer of the Cotton King's mummy bundle revealed in detail at nationalgeographic.com/ngm/0205.
 AOL Keyword: NatGeoMag

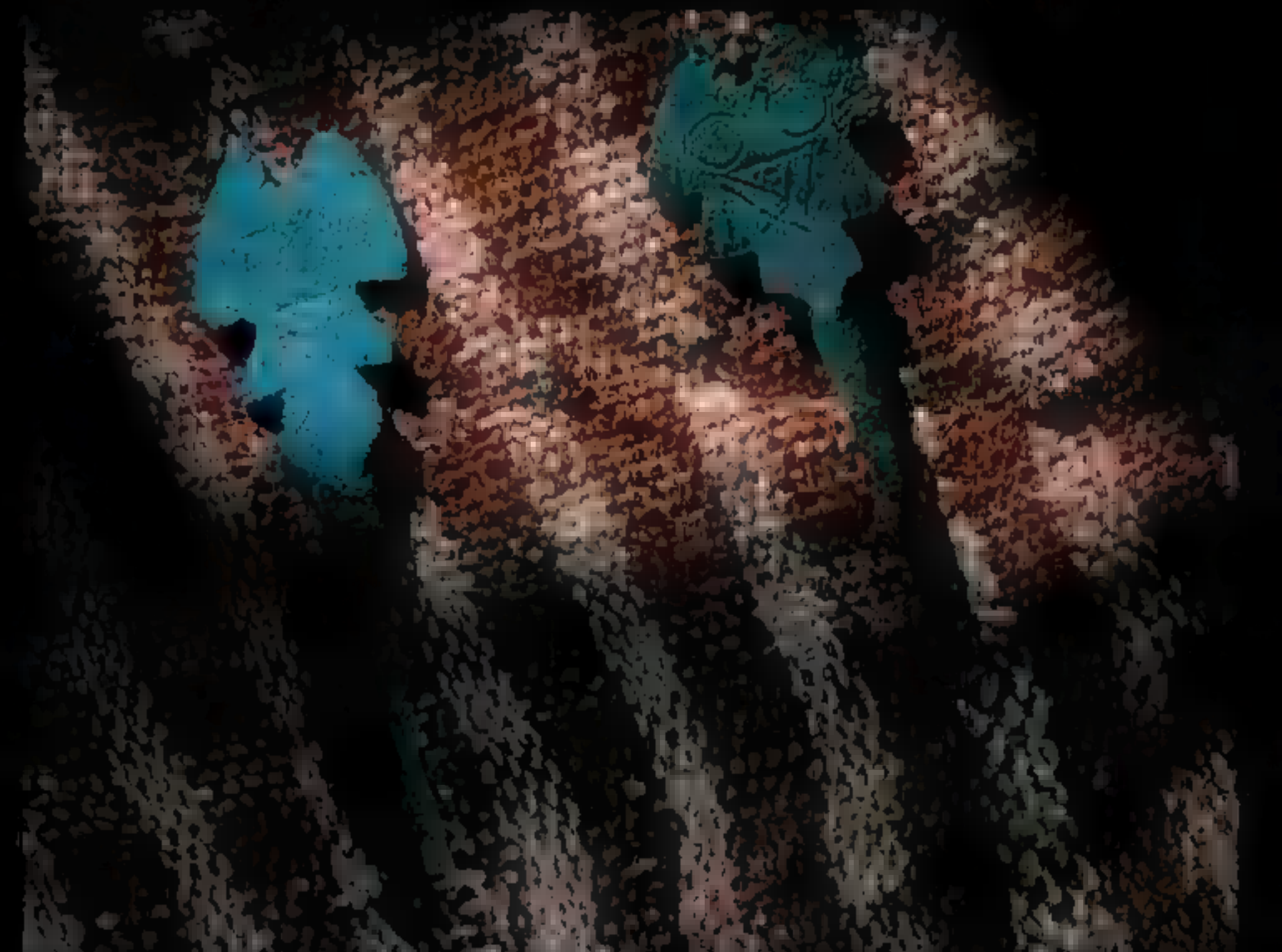
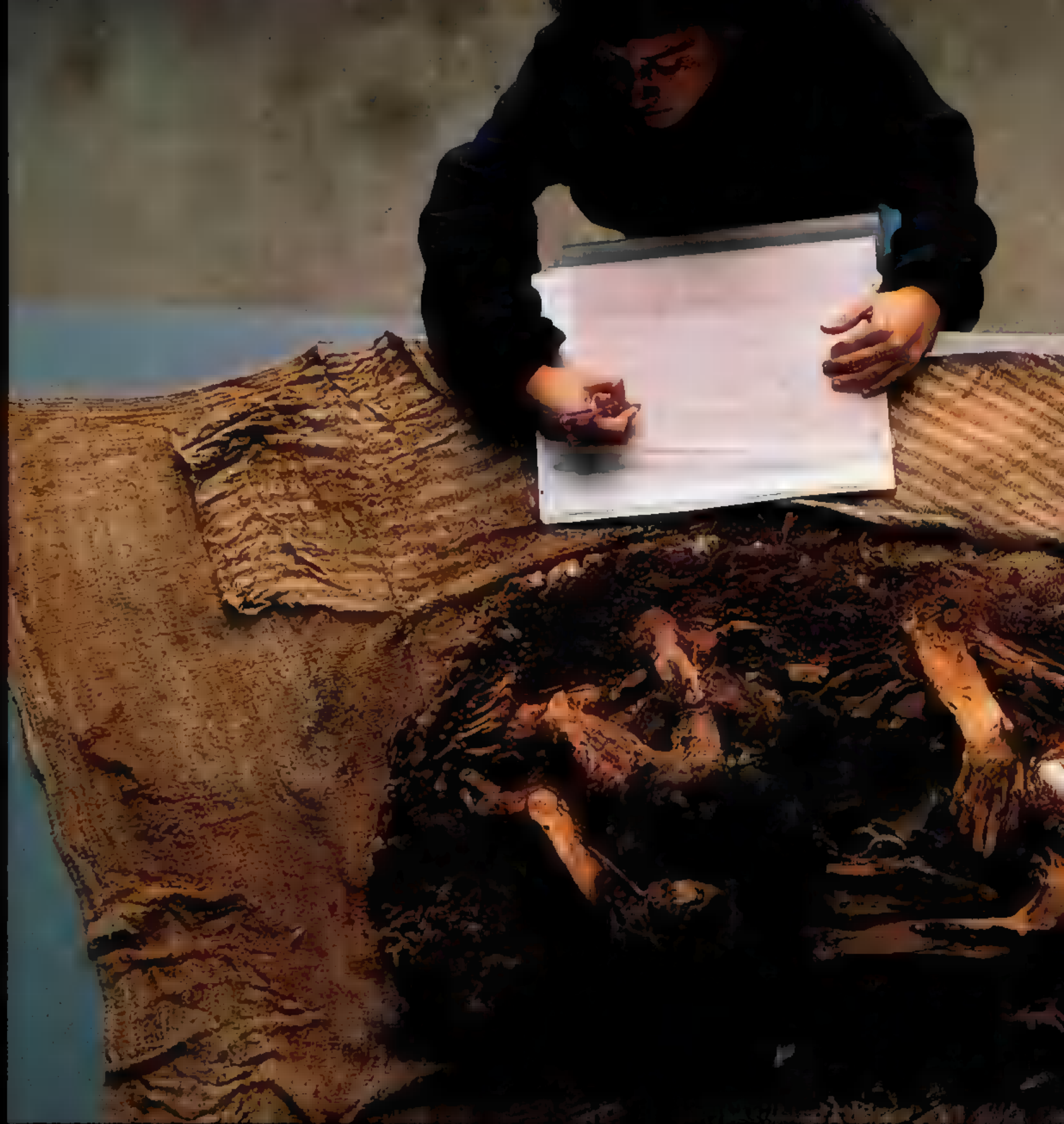
False heads, which likely give mummies a more human form, characterize the bundles unearthed at Parachuco. Some bore masks or wigs, but their false faces were left blank.

- Baby
- Feathers
- Cottonseed layer
- Mace
- Guinea pig skin



- Corn
- Sandals
- Sling
- Gourd
- Spondylus* shell
- Lime
- Silver tweezers
- Comb
- Coca leaves
- Sweet potato
- Bean pod

ART BY JOHN DAWSON (ARTIST) AND JOON WALES (RIGHT)





Bound for the Afterlife

The Cotton King's leathery hands (far left) clutch fabric, a shell, and a chalky ball of lime made from an aggregate of minerals. Locals today still chew lime with coca leaves to extract the stimulant within. The mummy is cleaned and sketched (left, top), finally free of cotton and most of the 170 items buried with him. These artifacts include (bottom, left to right) corn, peanuts, sweet potatoes, beans, and a gourd filled with lime powder; silver and copper *tupus*—pins to hold a garment closed; and a wooden comb with tines from a spiny plant and silver tweezers, black with corrosion. A figure (above) unearthed nearby still decorates the handle of a ceramic vessel.





Textiles for Eternity

Peruvian **weavers** were masters of **color**. An elaborate headdress with **ostrich** bird feathers and **long** **hanging** **ribbons** had a **front** **and** **back** **and** **two** **earflaps**, and a **long** **and** **wide** **shawl** draped down the back of the wearer, they would have been a person of high rank. **Wool** **and** **cotton** **woven** **bags** **and** **bags** was a **variety** **of** **colors** **and** **fabrics**. **Woolen** **clothing** **also** **cloaked** **the** **youngest** **and** **oldest** **for** **us** **the** **saddest** **and** **most**. Nearly half of those **buried** **were** **children**, who **were** **buried** **from** **infancy** **and** **were** **valuable** **to** **the** **family** **tended** **at** **death**, both **young** **and** **old** **were** **well** **equipped** **for** **the** **afterlife**. □

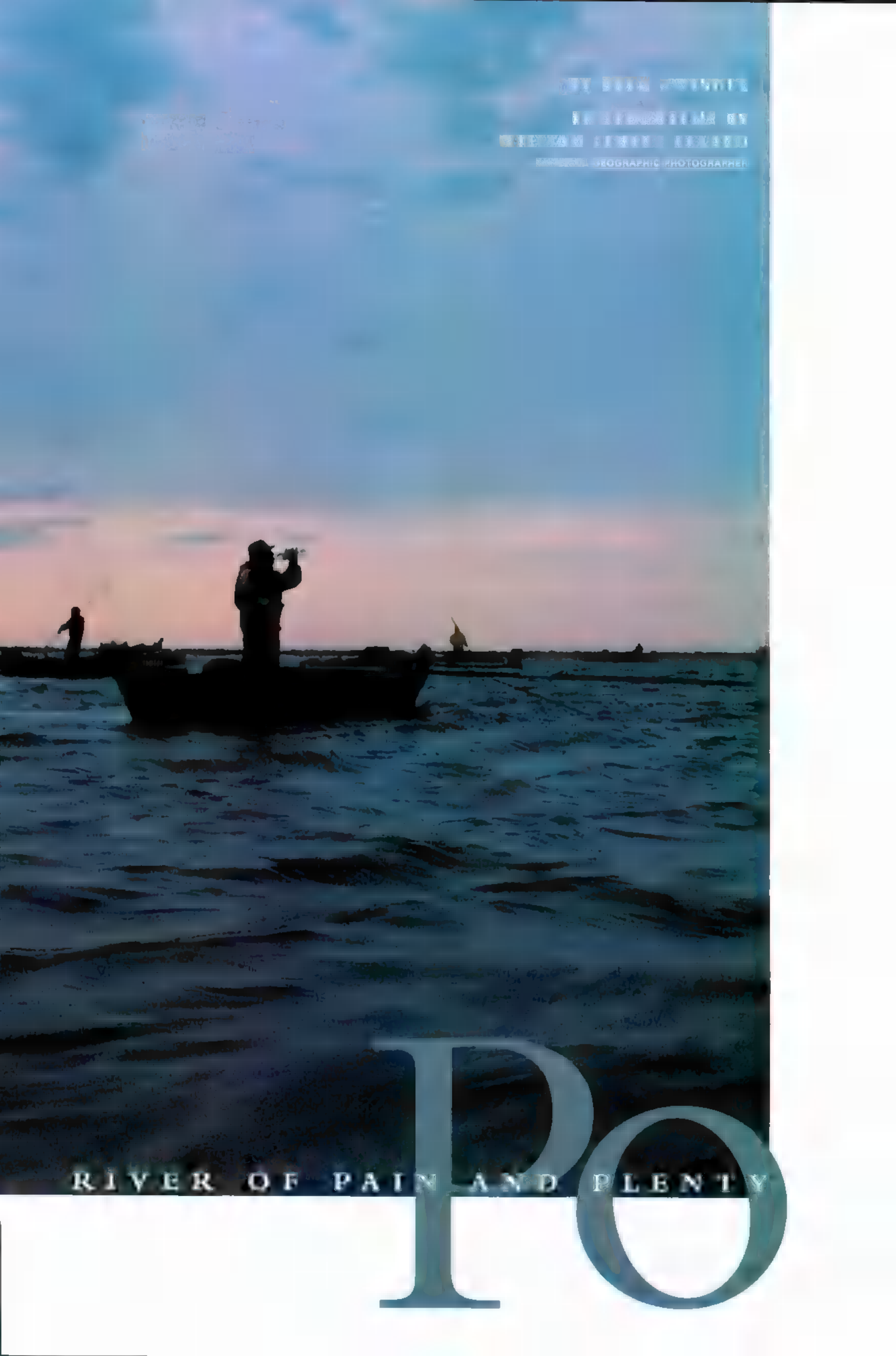




Wed to the river's ebb and flow, clammers harvest the "gold of the delta" where the Po meets the Adriatic. Dark and powerful, sinister in flood, Italy's longest river brings both peril and prosperity to those hardy enough—or lucky enough—to live within its reach.

THE RIVER OF PAIN AND PLENTY

NEW YORK: NATIONAL GEOGRAPHIC SOCIETY
A NATIONAL GEOGRAPHIC FILM BY
STEPHEN STREIBER
NATIONAL GEOGRAPHIC PHOTOGRAPHER



RIVER OF PAIN AND PLENTY

PO





BICYCLE BELLS signal rush hour in Ferrara, the medieval town turned Renaissance star under the powerful Este dynasty from 1260 to 1598. The walled city is one of several centers on the river that brought fortune and influence to the Po Valley.





“WE PRACTISED INTIMACY,” wrote Henry James of an Italian café afternoon, “an intimacy so much greater than the mere accidental.” Wine and words flow like the nearby river at Caffè Elena, a favorite haunt of Turin’s writers, artists, and soccer stars.





T A K E A P I N C H of sun, a sprinkle of rain, cool nights and warm days, a vineyard of well-drained Piedmont soil, and an ancient tradition around the hilltop town of La Morra. Age in oak barrels. Yield: vintage Barolos and Barbarescos—some of the best red wines in the world.

Attilio Formigoni knows the river better than he knows his wife. Thirty-eight years of navigating the sinuous, cunning waters of the Po, now finally as captain of his own tourist boat, the *River Queen*, have taught him how to read the waters in all of their languages of sound, color, movement. "I never tire of it," he said as he gazed out into the luminous summer night, the heavy boat sliding slowly with the current. "The Po has the beauty of change; that's its fascination. All this changing is what keeps your love alive."

He hardly glanced at the sonar screen that showed the shifting depths and shapes of the river bottom. "This is a river that requires experience," he explained. "First you learn it by eye. If you don't know what you're looking at, all this radar and sonar doesn't mean a thing." He talked about the hazards of navigating, about low water and floods and whirlpools and sandbanks, not to mention the expenses of buying and maintaining his boat. Why has he kept at it so long? "For passion," he replied without hesitating. "If you don't have passion, you'd quit immediately. Because to own a boat like this costs an eye from your head."

Love? Passion? When most Italians mention the Po, it's typically in connection with something bad, usually the ever more frequent spring and fall floods that tear away riverbanks and bridges and lay waste to fields and towns. The more fabled Arno and Tiber may have more glamour; the Po's fascination lies in its power, a force that inspires more fear than love. The Po exists in the national consciousness as something vast and potent beyond its dimensions, which some of the greatest Italian directors such as Visconti, Rossellini, and Antonioni recognized by making their finest films on or near it: *Ossessione*, *Paisà*, *Gente del Po*. "For most of us," said a man in Cremona, "the Po has always brought death, fatigue, danger." Drownings, destruction, grief. "The terrors which the Po has left in our blood," author Gianni Brera called it. *Al Po al dà 'e 'l tos*, they say: The Po gives and the Po takes away. Not for nothing did its earliest peoples call this vital, unpredictable stream *il dio Po*: the god Po.

Although the Po is known to every Italian schoolchild as the country's longest river, at 405 miles long and 1,650 feet across at its widest point it's a mere rivulet compared with the Nile or the Yangtze. But size is not the story. The Po's waters, fed by 141 tributaries draining a catchment basin of 27,000 square miles, created the Val Padana, the plain that stretches across northern Italy from the French border on the west to the Adriatic Sea on the east. More than 16 million people—nearly a third of all Italians—live in this fertile expanse, some of the most heavily cultivated land in Europe. Their settlements range from Turin, a major manufacturing town and headquarters of Fiat, the automotive conglomerate, to some of the country's most beautiful and historic towns: Piacenza, Pavia, Cremona, Mantova, and Ferrara. *Il grande fiume*, the great river, is clearly worthy of respect, if not, it would seem, affection.

Still, for everything the Po may have done to man, man has done at least as much to it. Nearly 25 percent of the land along its banks has been denuded of natural vegetation to make way for sterile plantations of poplars harvested for cellulose; the river is dammed for hydroelectric power and tainted by agricultural and industrial chemicals, to say nothing of the daily effluent from Milan, a city of 1.3 million—with no sewage treatment plant—situated on two of the Po's tributaries. (Ironically, another city of its size reprimanded by the European Environment Agency for its sewage problem is Brussels, seat of the European Union.) The illegal gouging out of 33 million cubic yards of sand and gravel every year for construction has left huge holes in the riverbed, some of its natural meandering curves have been straightened to aid navigation, and more than half its total length is immured by man-made earthen embankments called *argini* that protect towns and fields, all of which have only made the Po's floods fiercer and more disastrous.

Yet beneath the incessant recitation of the river's real problems you can discern murmurs of love. They are like the wordless voice of the river itself, a sound that is half water,



Spreading between the Alps and the Apennines, the Po basin covers a quarter of Italy. The river itself, whose earliest name, Bodincus, meant “bottomless,” stretches 405 miles from source to sea.

half wind, or like the tiny ripples that are caused not by the breeze on the surface but by the undulations of the hidden riverbed far beneath. “There are people who are rooted in the Po,” one man told me, “so that even if he hits you, you turn the other cheek.” This passion for the river is the story.

Because I am naturally contrary, I began at the end, in the delta. The Po finishes on the country’s northeast coast, ramifying into five branches spreading across an area that was once marshy wetlands. Once it was farmland owned by Venetian noble families; in the early 20th century it was as poor as the Mississippi Delta, the scene of desperate struggles of the farmworkers against the landowners.

Though still a seemingly remote corner of the Veneto region, today the Po Delta comprises some 170 square miles of rich bottomland, among Italy’s most valuable at \$10,000 an acre. Since the 1950s, land reform, farm subsidies, and generally higher national prosperity have mitigated the poverty. What hasn’t changed is the evidence of generations of heroic toil to reclaim the land from the river—the pumping of water, the carrying away of soil in infinite processions of wheelbarrows. Although in many places upriver sedimentation has raised the riverbed above the level of the surrounding fields (“where the birds live lower than the fish,” the country people say), here in the delta almost half the fields are between six and twelve feet below sea level, due partly to offshore methane extraction. Here the argini must defend fields of corn, wheat, and rice from both river and sea.

One of the richest crops harvested in the delta doesn’t come from the land, but from the water: fish. The coastal area is pocked with “fish valleys,” aquaculture basins full of gilthead and sea bass. And there are shellfish: If you drive along the embankment that flanks the

broad brackish bay called the Sacca di Scardovari, you see nothing but cultivation stretching to the horizon—on one side, emerald green swaths of rice and on the other, the iron gray lagoon waters that nourish clams and mussels.

Mario Moretti, 53 years old, began clamming when he was 30. Now living in the hamlet of Gorino Sullam, he is small and tough, with a heavily lined face lightened by a sweet, candid expression, and a back whose warranty has long since expired. “You’ll see how fatiguing it is,” he told me. “I think it’s work you can only do for ten years. After that you can’t do it anymore. If somebody starts at 15, by the time he’s 30 he’s ready to be thrown away.”

We went out one chilly April morning at four, an hour dictated by the waiting trucks more than the unsuspecting clams. The *garbin* was blowing, the strong, damp southwest wind that Mario curtly called the fog wind. The predawn darkness was already blurry. We motored down the branch called the Po di Gnocca, cutting its glassy smoothness with our prow, and out into the choppy open waters of the Sacca di Scardovari. Mario threw out the anchor and got his gear ready, but he wasn’t hopeful.

“Too many waves,” he muttered. “We really got a bad, bad day.” The whole world was gray: gray water, gray faces. The only spot of color was Mario’s red Ferrari baseball cap.

He started to dig for clams, using a long metal pole with an end like a metal rake with a net bag attached. Bracing this pole against his right shoulder, he dragged it across the bottom with powerful jerks, half-bending and straightening with each yank. When he had done this for several minutes, he hauled it up with a simple winch and emptied the clattering clams onto the foredeck to be sorted by hand. “It’s the law to fish like this, because it’s better for the environment,” he said. Boats in nearby lagoons that illegally use what amount to motorized vacuum cleaners obviously take more with no effort, “but they destroy the bottom completely,” he said. “In one day there’d be nothing left here.”

As the fog dissolved into pale sunshine, I could count some 50 boats out clamming. In the gray horizonless expanse it felt as if we were at sea. But the Po is never far, and when it floods, it plays havoc with the clam and mussel beds. “Not because the influx of fresh water changes the salinity so much as to damage the clams,” Mario explained, “but it shifts the land. That tongue of land over there is more open now, and the water goes more out to sea. The flood of October 2000 silted up the clam area, and clams really need the tidal flow. They don’t die, but they grow too slowly.”

We sorted and bagged the clams and took them to the weighing station operated by his cooperative. A mere seven pounds, as opposed to his usual twenty. But apart from the beads of sweat, he was as serene as when we started four hours ago. “I was born a fisherman, I live like a fisherman,” he said. “If you’re born an engineer, you live like an engineer. He eats out 15 times a month; I eat out once a month. I have a wife and a son, and my work. Even if I earn less compared with other work, I wouldn’t change, no no. It’s the tranquillity.”

Carolina Marisa Occari is 75, gray-haired and slowing, and what she harvests from the Po is beauty. Marisa is an artist, and though she now lives in Ferrara, in the delta’s



southwest corner, she was born on the river and has spent her life making etchings of it. We sat talking in the sunny brick courtyard of Ca' Cornera, a farmhouse outside Porto Viro now converted to a restaurant and inn. The owners, Paolo and Antonella Gasparetto, have turned the old granary into an exhibition space for artists such as Marisa and others who devote themselves to depicting this subtle, not immediately beautiful landscape.

"When I'm by the river, I forget every tension," Marisa said in her quiet, clear voice. She was wearing a long necklace of amber beads, a gleaming reminder of the myth of Phaeton. The Greeks believed that he lost control of his father Helios's chariot of the sun and plunged to his death in the waters of the Po. His weeping sisters became the poplar trees along its banks; their tears were transformed into drops of amber. The Greeks called the Po Eridanus and sailed to its mouth to trade for products from northern Europe, including Baltic amber.

Marisa had brought her tools, ready to work; we got involved in talking instead. But I



M O R N I N G S O L I T U D E *along the river belies the usual bustle in the historic section of Turin, the largest city on its banks. A mix of baroque splendor and urban sprawl, the city straddles an ancient ford of the Po.*

had seen photographs of her sitting on the embankment under an umbrella, transcribing the panorama directly onto the metal plate, “from life,” a most unusual thing for an etcher to do. “When I was growing up, I had to bicycle to school, and the road was on top of the argine,” she was remembering. “There was a whole rich life in the *golene*”—the flat stretches of bottomland between the river and the embankment, now mostly under cultivation—“you can’t imagine. Shepherds who passed with their sheep. Men fishing, people sunbathing. The *golene* were a whole world.”

Marisa’s black-and-white riverscapes seem full of space and movement, massive rustling trees, swelling white clouds. The Po doesn’t call for many colors, but it does call for shapes and volumes. “It’s mysterious, this flux, this going and coming of the water,” she went on. “Here we don’t have immense rivers like you do in America. For us this is the big river, so majestic. You feel that you have to respect it. You feel that you can’t dominate it. I have love for it, love and respect, and I feel its greatness. Our grandparents always presented it as something dangerous to us. But to live in contact with nature gives you the



force to face your problems, even if they remain.” Here was another who could love it and revive her spirit in its liquid pulse. “Water gives me a sense of grace, unlimited space, of peace,” Marisa said. “It’s a symbol of life to me.”

Life in the central part of the Padana plain (the term comes from Padus, the Latin name for the Po) is firmly connected to extravagant stretches of farmland, dotted by tiny villages and a few larger towns, most now hidden behind the river’s embankments. Seen from the water, the only hint of the land beyond the green ramparts is the occasional church spire or bell tower; from the land the river is invisible, restrained like a sort of caged creature. This earthen bulwark has blocked the casual encounter and the distant fond glimpse, and forces land and water into an oddly confrontational stance.

This is the spacious homeland of some of Italy’s more delectable specialties, from the



Agriculture and industry drive the Po basin economy—often at the expense of the river. Plantations of poplars, cut for pulp near the delta (above), have displaced natural habitat. Fiat, the Turin-based auto giant (left), has been a catalyst of economic and social change, luring many southern Italians north. Yet industry contributes half of the pollutants entering the Po, agriculture a third.

onions of Boretto to the Parmesan and prosciutto of Parma, from the wines of the Oltrepò Pavese near Pavia to the rice from the fields of Vercelli and Lomellina. Italy is Europe's leading rice producer, thanks in part to water from the Po and its tributaries that irrigates this territory through a network of canals.

The main stretch of river, from Ferrara to Cremona, was a major shipping artery until the 19th century, when trains and then highways drained its traffic. Cremona is not the only working port on the river—Ferrara and Mantova are also active—but Cremona is the last point that shipping can reach, and a hydroelectric dam just beyond has put an end to further commercial navigation. The city, of course, is more famous as the home of the world's greatest violins. Yet some Cremonese continue to “live” their river: for diversion.

Slim and still athletic, Fulvio Ghisolfi has been a national kayak champion, and he spent years training on the Po from his club, the Canottieri Baldesio. Before that his childhood summers focused on Ponticello Beach, a broad apron of river sand where his father ran a small refreshment stand. Up to the 1970s the Cremonese would come to the river in droves to swim, sunbathe, and escape the suffocating heat of the flatland summer.

The Po has since washed the beach away, and Cremonese mothers warn their children that the river is too polluted for swimming. But Cremona still boasts five rowing clubs on the Po, with a combined membership of almost 15,000. Even though only a small percentage of those actually row, on a bright Sunday morning in June the river was full of boats ready to row the nearly 35 miles from Cremona to Casalmaggiore in the annual Vogalonga, or “long row.” I was there, gripping my oar along with three other rowers: Ettore Pigoli, Palmiro Ardovini, and Fulvio himself.

There was a time until maybe 30 years ago when many people rowed, from fishermen to Sunday-afternoon families. Our boat was an aluminum replica of the traditional wooden riverboat of the Po, a slim, flat-bottomed lozenge that we rowed standing up, facing forward, exactly like Venetian gondoliers. At nine we set off, a multitude of colorful boats that quickly separated and strung out on the broad brown waters. It would take us some four hours to reach Casalmaggiore, and the sun was already hot. We rowed steadily but not rapidly; the water was unusually high for summertime and bore us along at an unhurried, confident pace. Cornfields and poplar plantations lined both sides, and we slid through floating white clouds, the brilliant, majestic cumulus reflected in the glassy water.

We stayed toward the middle, sometimes letting the water shift us at an oblique angle; near the shore we would have found water twisting and turning back upstream against us. All this immense plain was once under the ocean, gradually exposed as the Adriatic retreated and then gradually augmented by the sediment carried down from the Alps by the Po. Paleontologists exploring the riverbanks still find the remains of prehistoric bison



and deer, Bronze Age pottery, and even ancient human bones. Today, as we rowed along in the breezy sunshine, the river seemed almost playful, willing to sparkle and let us slide over its sleek flanks. The water and sky were the same hue, a pale opaque tint that was more glare than color. The banks were deep and green and mute. Though the river seemed silent, there was something in the sense of it pulling me along, giving occasional small shivers of waves, that hinted at its strength. “The day the flood was near my house,” another man from Cremona had told me earlier, “it seemed that the river was saying, ‘Look how powerful I am—make your decisions accordingly.’” Now, even when all was well, I sensed what he had meant. When we reached Casalmaggiore, the sun had begun its slow daily fall toward the Po. The leaves of the white poplar trees were bright in the wind.

The poplars have lost their mythical meaning and are now a mere commodity, supplanting a number of native species that once graced the riverbanks. Here in Casalmaggiore the



Traditions abound along the Po, though not without a modern twist. During the Palio of Ferrara—a 700-year-old festival centered around a horse race—costumed locals (left) parade to pay “homage” at Este Castle. In Cremona (above), home of the Stradivarius violin, artisans from around the globe learn to build instruments the way the maestro did: by hand, one at a time.

Consorzio Forestale Padano, a private consortium working with contributions from the EU, has been busy for seven years restoring the poplar to the riverside environment. The ubiquitous stark plantations of commercial poplar trees have created problems, not only by altering the natural habitat for other flora and fauna and by the runoff from their twice-yearly pesticide application, but also by posing a risk when the river floods. Because they are harvested in eight-year cycles, the commercial trees have no time to sink the broad, deep root networks of long-term native trees, and so the surging water can tear them away and hurl them downstream where they either damage bridges and buildings or jam up with other debris to block and derange the waters further.

Emilio Dell’Asta, the 55-year-old president of the Consorzio, has always known the river. “We swam in the irrigation canals that brought the Po to the fields,” he remembered. “In those days they were nine feet deep; there were a lot of fish in them.” He took me along the riverbank and explained the reforestation projects in the golene. “We’re planting trees typical of the past,” he said, “so that we can create a kind of park. We take the plants that always grew naturally here—ash, alder, oak, poplar, sometimes native walnut and wild cherry. So far we’ve planted 120,000 trees.”

He showed me a long riverside alley formed by 500 stately elms—250 on each side—that they had also placed there. It was shady and cool. This is the only area in northern Italy where such a restoration project has been undertaken, but the Consorzio is planning others. “If there are 1,000 hectares [2,500 acres] of golene along the Po from Cremona to the delta, it will take us 20 years,” he said, “but this is what we’d like.

“And now we say, when it’s all done, what will it be for?” he continued as he gazed at

the sheet of water glittering behind the shaggy undergrowth. "Build observation towers with binoculars for children to come and bird-watch. Plan trails with signs for teaching about plants. We'll have picnic places where families can come." He stopped short of mentioning shepherds and sheep, but I remembered the "rich life of the golene" that had so enchanted Marisa Occari. "Living" the river, I often heard it called, is a recollection of the not so distant days when the river supported generations of families, not only fishermen but also reed-gatherers, brickmakers, and millers whose mills floated in midstream to catch the strongest current. To "live" the river is an idea that needs to be replanted too, and here it seemed to be taking root. "The Po was a great accomplice of adolescence," one man recalled. "The first girlfriend you had, you took there." Maybe it could happen again.

The Po from Cremona upstream toward its source passes through increasingly lovely scenery, green hills draped with vineyards reaching for the snowy Alps beyond. But somehow the river becomes less romantic. For although these hills are the fatherland of some of the country's most prized products—Alba's white truffles and the noble red wines of Barolo—here the river basin is thick with industry, hundreds of small businesses producing everything from paint to leather garments, luxury chocolates to the famous Borsalino hats. These enterprises account for much of northern Italy's impressive prosperity, but they can also pollute. Meeting all the legal environmental standards is prohibitively expensive for companies with only a few dozen employees, and that includes farms. The river is polluted to varying degrees along its path by industry, agriculture, livestock, and urban effluent—especially the sewage brought by the tributaries flowing through the city of Milan. Still, Ferrara, on the edge of the delta, takes nearly 80 percent of its drinking water from the Po.

The mountains beyond Turin are where the floods start. Between the melting snow and glaciers in the Alps in spring and the torrential autumn storms that boil up over the Apennines, the overflowing tributaries pour their waters into the Po, which swells as it races downstream. Improvements in forecasting and monitoring rainfall are saving lives: In the 1994 flood there were 64 deaths, but in the much larger flood of October 2000 there were only two, because the alarm was given in time.

This is encouraging, but unfortunately it doesn't imply a more peaceful coexistence with the Po, because people are still determined to fend off the river from their houses and fields. Maps of the Po hundreds of years ago trace a twisting stream full of unbarricaded oxbows, which both slowed and dispersed floodwaters in a slow and equable manner. Today the river is shaped, and behaves, more like a bobsled run. "When the Po needs to expand and covers the houses," said Luigi Ronda in Cremona, who publishes an elegant magazine called *Il Grande Fiume*, "it's clear that it's humans who were wrong."

Because half the Po's riverside settlements are rated as being at high to very high risk from floods, the Po Basin Authority has proposed a flood-control measure by which



the river would be allowed to overflow farmland in order to lessen damage to nearby towns. Naturally the rice farmers of the Vercelli area have protested. And it's not just homeowners and farmers who demand protection from the river; the Po Valley is, like most of Italy, home to uncounted works of art.

Al Po al v  'ndee' cal v , they say—the Po goes where he wants. Not if his neighbors have anything to say about it.

Professor Roberto Passino, the secretary-general of the Po Basin Authority, is an engineer, but he explained what's happened to the Po in distinctly unscientific terms. "It may sound strange if I say this," Passino said, "but there isn't a love for the river. If there had been, this sentiment would have fostered more harmonious development. It would have inspired resistance against abuses." If I'd heard this several months earlier, I might have believed him, but by now I'd met Attilio Formigoni and Marisa Occari, Fulvio Ghisolfi and Luigi Ronda. The river is deeply loved by those who know it; it's just that they're



N E W L A N D born of river sediments adds 200 acres a year to the fertile Po Delta, which has been drained for cropland since Roman times. Rice fields and hunters' cabins jut into the Adriatic near the Sacca di Scardovari.


outnumbered by those who don't. "I bought a house on the banks of the Po to see the sunset, and my friends regard me as a cretin," one man admitted. "But I think the Po has something of the sacred to those of us who live near it."

Passino may not go that far, but he did say this: "We need to give back to the river everything that we have taken from it in past decades. There are some things that can't be replaced. But we need to stop the degradation. We need to have the courage and strength to resist construction of more embankments that create more problems for the river. We need to distance the settlements from the river—houses, factories—and not permit roads and railways near the river. Limit the extraction of sand in order to protect the river." Protect the river? What a lovely, and unusual, thing to hear.

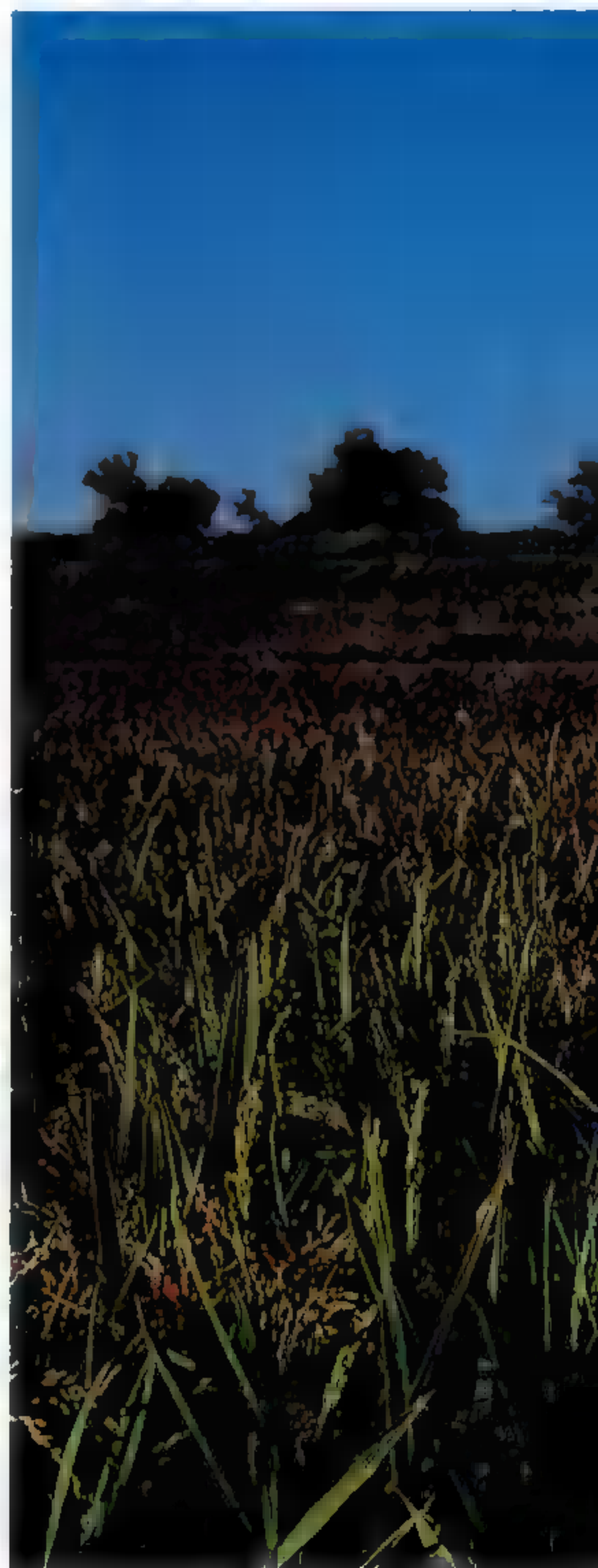
Westward from Turin, as I drove the curving narrow road up the Po Valley toward the source, I noticed the farms and villages were smaller. There's more forest, less traffic; less corn and more fruit trees. I arrived at the Pian del Re, the upland bowl where the Po rises from the flank of Monviso, late on a June afternoon. A herd of sheep were moving slowly up toward the even higher valleys, followed by their shepherds and sheepdogs. I parked and walked over the grassy meadow to a tumble of rocks half-shielded by a grimy carapace of old snow. The stream pouring out of the darkness was the Po.

Clean, muscular, intrepid. I sat on a rock and looked at it. I had known it would be small, but the heavy bulk of the mountains, the sharp air, the stillness broken only by the boisterous rush of the water and the distant whistling of marmots all gave it a power just as strong as, though completely different from, the ponderous flow downstream. Here the Po was supple, full of energy, leaping over stones and down the mountainside impatient to meet its friends and enemies: Sesia, Lambro, Adda, and Oglio, Mincio and Taro and Ticino, all the tributaries that were also hurrying to meet it. The broken tablet on a boulder says simply *Qui nasce il Po*, "Here the Po is born," but it struck me that the Po was really being reborn. When you see the river downstream, heavy and muddy and old, and then see it here, glittering and blithe, it takes an effort to realize that it's all the same water, having made its seaward journey countless times, always returning to its mountain cavern as rain, falling like Phaeton to begin again.

In the distance I could make out the heat-veiled plain below, waiting to receive more water, and more earth. I could imagine what lay ahead. Not just pig farms and cement barges, but the languorous dawn stretches echoing the hidden cuckoo's call, and the dark twilight reaches of the delta, where chill shadows are fragrant with the fugitive scents of wet wood and blossoming lime trees, where slow waters press against the tide, and black trees hint at prehistoric figures creeping down to the water's edge to wash some unnameable object.

 **OUR WEBSITE**

Erla Zwinge and Bill Allard share their experiences of Italy's great river at nationalgeographic.com/ngm/0205.
AOL Keyword: NatGeoMag



More than that, people are waiting, in whose blood the Po hasn't left only terrors—the people who love the Po, like Fabrizio Boscolo, not yet 50, who was born in a cabin on an islet in the delta. “Till 1967 I drank the Po water,” he had told me as we motored slowly among the reedy mudflats. “My father made me a drink of wine, Po water, sugar, lemon, and lots of ice from the fish market. We would eat duck broth with beans and eel in tomato sauce. We lived by fishing, and my mother made the pasta with duck eggs.”

“I think we've passed the worst moment,” Luigi Ronda reflected. “I see the river as a point of union. It's like the sky.”

But it was Giorgia Ghisolfi, Fulvio's grown daughter in Cremona, who unwittingly gave me the clearest sign that all might yet be well for the great river. Her mother, still astonished, told me how Giorgia had come back from her vacation one summer on the coast of Puglia. There the sea is famous for its crystal iridescence. Yet the instant she glimpsed the lazy, turbid waters of the Po, she was ecstatic: “My ocean!” she cried.



SICKLE IN HAND, a seasonal worker culls weeds the hard way on the Po plain, Europe's leading rice-producing region. Such hand labor is now rare on the plain, one of the few areas in Italy level enough for large-scale farming.





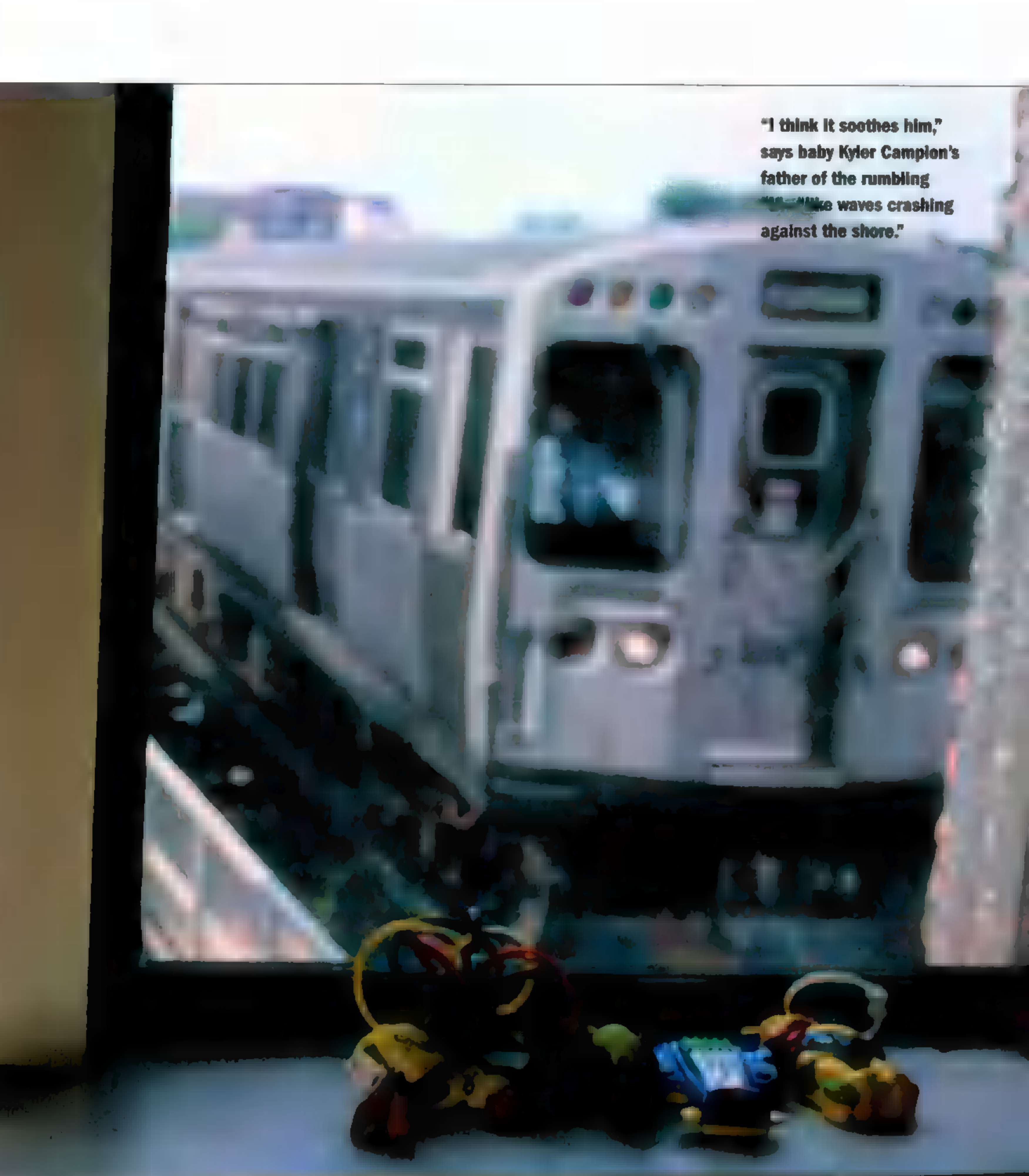
SUNBATHERS WAIT for rays on a beach near Polesella. Though it may look benign, the Po is ignored at one's own risk. New development and dikes have led to devastating floods, which are increasing in frequency, while pollution has closed bathing beaches in some areas.





A P R I C O T B L O S S O M S shower a lane along Turin's Valentino Park, where, according to folk songs, college boys meet factory girls for trysts by the river. With its floods, fog, and spectral beauty, the Po—like a bittersweet romance—tugs at the heartstrings of Italy. □





"I think it soothes him,"
says baby Kyler Campion's
father of the rumbling
"like waves crashing
against the shore."

60614

Life by the Tracks

BY SHANE DUBOW

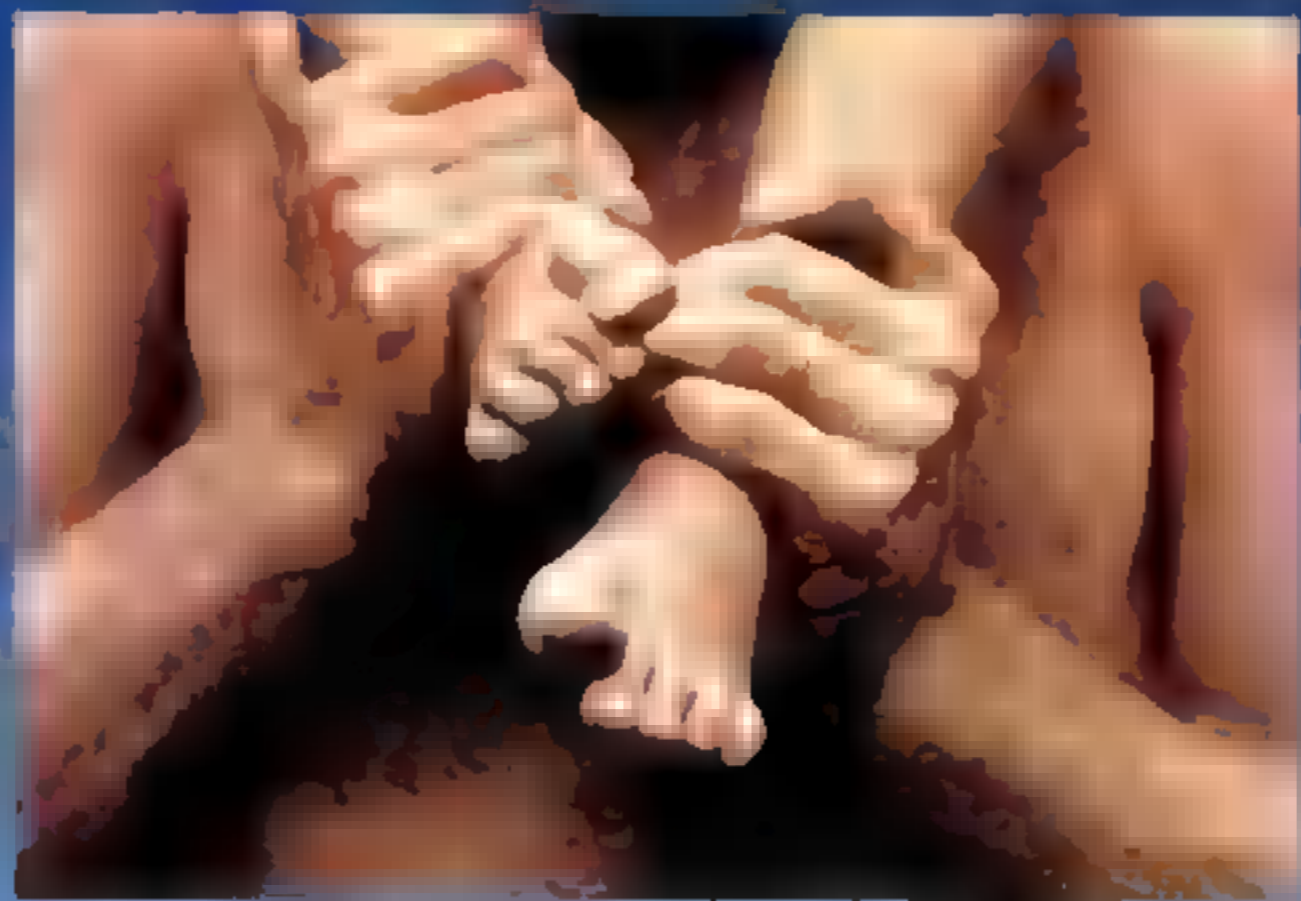
PHOTOGRAPHS BY NINA BERMAN

It seems so improbable, that a heavily urban rail line could feel so cozy and unhurried, so curious and human, as if built more for neighborly voyeurism than transit. And yet, for a fare of just \$1.50, a ride on one of the last of Chicago's elevated train lines grants these trackside views: a young woman in a pink hash-broth steaming espresso; a young man in trim khakis feeding a baby; a great many more waiting on the platforms, digging into newspapers, checking on their Cubs; and then a long, slow blur of flower-bed gardens and second-story decks, all passing so close that rider and resident might intimately converse—or even smile—were it not for the heat and rush of the “L.” This is Chicago's most celebrated mode of transit, a 110-year-old relic whose redefining downtown run has helped revitalize the neighborhood of Lincoln Park.

You get used to the noise, John and Wiley Kelly are telling me one afternoon, as the first de-lousing commuters trickle into their trackside Webster Avenue pubs. And indeed, a quick scan of the room, all aging sports photos and dark wood trim, reveals an unobtrusive small aversion to the recurring ill. One of the neighborhood's three train stops, at Armitage, lies two blocks south; another, at Fullerton, two blocks north. In a 100,000-square-foot neighborhood, that's a lot

Lincoln Park's northern edge slumbers in darkness between the glow of Lincoln Park's skyline and a train streaking home.





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POPULATION: 65,474
MEDIA AGE: 31
MEDIAN PRICE OF ONE-BEDROOM CONDO: \$201,900
MEDIAN PRICE OF SINGLE-FAMILY HOME: \$372,000
NUMBER OF L STATIONS: Three
TIME BETWEEN RUSH-HOUR TRAINS: Three to six minutes
AGE OF THE L: 110 years and counting

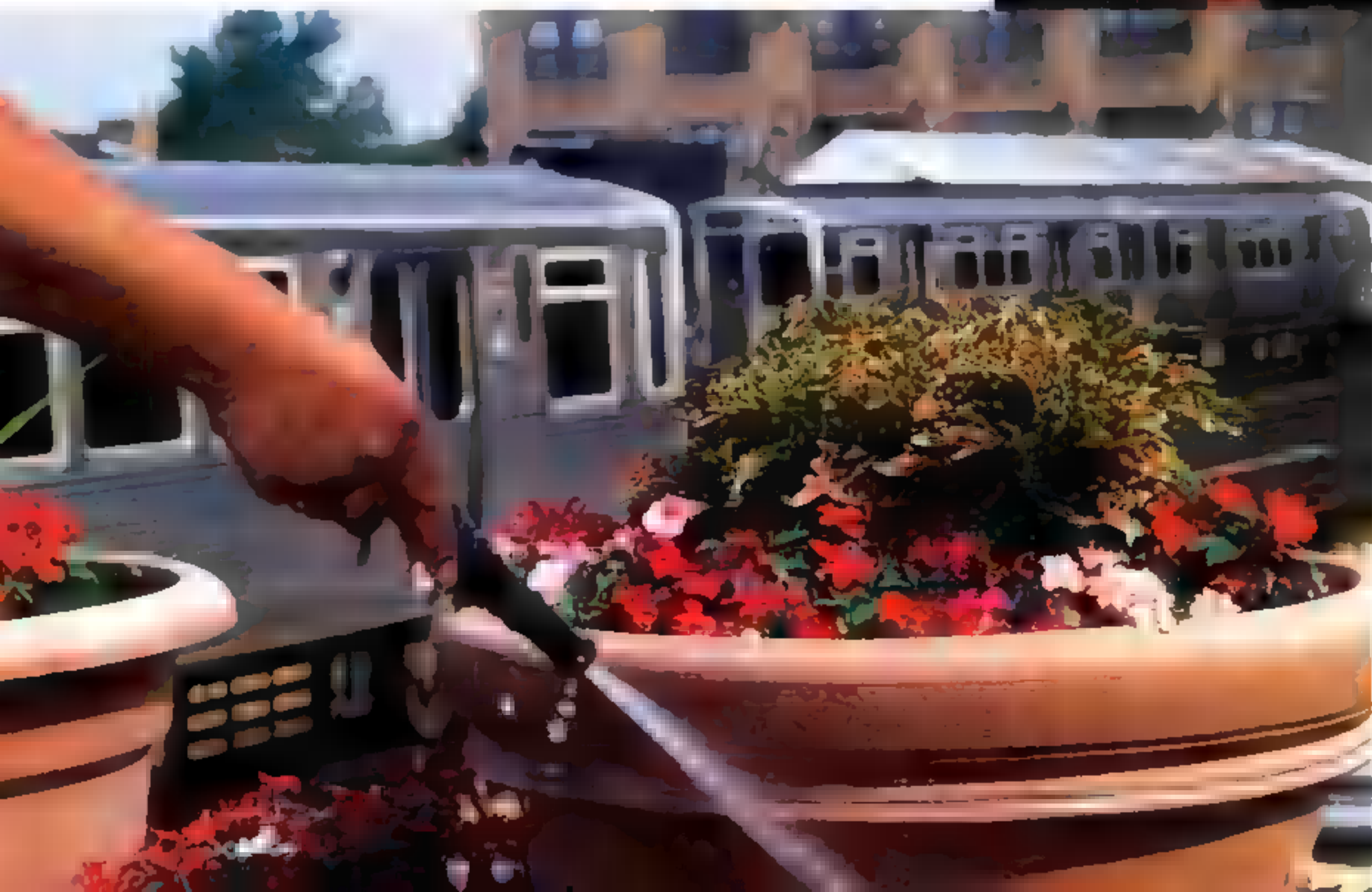
of stops. What you do here in between them, they say, whenever a train passes, is sip your beer, feel the vibrations, and think of what to say next.

“It’s a good place for first dates,” John says.

The story of how a few miles of old train line helped a tired neighborhood refill with cafés and cleaners, gyms and condos, landscaped yards and million-dollar homes goes like this: Out of the ash of the Great Chicago Fire of 1871, at the edge of where city living stopped and North Side truck farming began, a village sprang up. Built largely of two-story Victorian buildings, it soon featured an elevated train line. Boom times followed. German and Irish families settled in. Gangster life flourished. The Depression dawned. Kelly’s Pub, then called the L Tavern, opened after Prohibition, not far from where the FBI would gun down John Dillinger. Car culture came on. The suburbs thrived. The city crumbled. Rail travel declined.

It wasn’t until that same car culture choked the highways that more and more young people began to settle in rail-rich Lincoln Park. Between 1987 and 1998, for example, annual Brown Line ridership jumped 37 percent to 11.2 million. The young singles, like the recent DePaul University grads who live above Kelly’s and display an impressive collection of shot glasses in their den, come for the action, a tremendous concentration of bustling restaurants and post-frat bars. “It’s like being in college except with money,” is a refrain you hear a lot. The couples, like Greg and Julie Johnson who live in a renovated row house four blocks north, come to raise kids in a zip code that has come to be known as one of the city’s safest and most lovingly restored.

But isn’t owning an L-side home, especially with kids, a different sort of commitment from renting an L-side bachelor flat? Greg, an investment banker, shakes his head. We are seated in his tiny backyard, in the shade of the tracks, surrounded by children’s toys, a baby monitor, and a Border collie, Cassie, whose behavior may say less about trackside living than the weirdness of dogs. For the southbound trains, Cassie sulks under the deck. For the northbounders,



Players volley and gardens grow just a short lobe from the tracks. “People look forward to seeing my flowers,” says Pam Stosur, watering at left. “I like to add a little color to their commute.”

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Henry Alexander, 56
Retiree, Coach, Avid Gardener,
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she spins twice, barks, then bounces, feet first, off the side of the house.

"It's really not bad here," Johnson says. In the house the sound might equal the sound of a loud vacuum cleaner. Johnson taught his kids to cover their ears when playing outside. "When I bought this place, I got maybe a 30 percent discount," he adds. "Someday we might move to the suburbs, but you can't say enough about what it's like to have this transportation. It only takes me 25 minutes door-to-door to get to work."

To cope with the noise, some trackside dwellers double and even triple brick their walls. Air-conditioning muffles sound. One couple ordered closed-captioned TV. There have been apartments in which wall-to-wall carpeting was a literal concept. And then there are those whose relationship with the L has grown a tad more involved. One man stood a scantily clad female mannequin in his window, just for kicks. Another, the story goes, woke one morning unable to find his keys until someone waiting on the Armitage platform glanced in to point them out. Still another remembers the time a passing train slipped off the tracks, stranding dozens between Armitage and Fullerton. "We were talking to 'em out the upstairs window," trackside resident Tom Tiernan recalls, "making phone calls for 'em, calling their work to say they'd be late. When they got down, I think a lot of them went over to Kelly's and got free drinks."

"Yeah, I remember that," John Kelly says later back at the pub.

Polly Kelly nods. "That's how it used to be around here all the time," she says, "a real family place. In the old days people used to send their kids down to get a bucket of beer, and they'd bring their own bucket."

A train roars past. Polly Kelly quiets. Outside, through the open doorway, it's easy to see the streets backing up, long lines of gleaming vehicles, the exact sort of congestion that, were it not for the L, might grow to choke the neighborhood entirely. Instead, the L remains, and the neighborhood thrives on foot and rail traffic, and the only notable gripes seem to come from those who dream of rubber wheels.

"John is always making that joke," Polly says. "When are we going to get those rubber wheels?"

"I understand they're very quiet," John says.

Another train passes.

Polly Kelly rolls her eyes. □

MORE INFORMATION

ON OUR WEBSITE There's more on 60614 at nationalgeographic.com/ngm/0205. Tell us about **YOUR FAVORITE ZIP CODE** at nationalgeographic.com/ngm/zipcode/0205 or mail your idea to PO Box 98199, Washington, DC 20090-8199. E-mail: zip@nationalgeographic.com



Bissell Street bustles during the annual Sheffield Garden Walk. "This is a typical mood—there's not that anonymous city feel," says a former Chicago resident. "People love this place despite the train, or maybe because of it."

Final Edit



PO RIVER

Stolen Moment

It was during the Palio of Ferrara, a centuries-old festival celebrated in one of the former ducal capitals along Italy's Po River. Townspeople in period dress were parading into the cathedral to have their banners blessed, while NATIONAL GEOGRAPHIC photographer William Albert Allard wandered the church looking for situations that go beyond the obvious, that ask questions that may not have immediate answers. That's when he saw a couple waiting for the ceremony to end. "It was a very human moment," says Allard. "A young man and woman, possibly in love, with all the attendant highs and lows, sitting a few feet from a priest who was listening to someone else's problems." The picture gives us a clue about the Italian people, says Allard. "I don't think you'd see this in a Catholic church in Minnesota."

MORE ON OUR WEBSITE

Cut or keep it? Find out what tipped the balance for this photo at nationalgeographic.com/ngm/0205.

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KENYA

Daredevil Gastronomy

When in Kakamega, eat as the Kakamegans do

Photographer **Jim Richardson** says he's "always shied away from adventurous eating" while in the field. But visiting the western Kenya town of Kakamega while photographing an experiment in biotechnology for this month's article on genetically engineered foods, he was persuaded by a

market vendor to try a local seasonal delicacy: white ants, or termites (left).

"They're eaten either fried or straight alive," Jim says. He opted for the straight alive version. "You reach in, grab a handful of about seven or eight at a time, and pop them into your mouth." No, they don't taste like chicken,

he adds. In fact, they don't taste like much at all. "They don't have a lot of flavor, but they have a certain textural quality," he notes. "The lingering aftereffect is of white lacy wings stuck in your teeth."

Jim says he began work on the assignment "totally information-challenged," knowing little about the biotech wars. He came away from it fearing not the effect genetically engineered foods would have on human health but their potential dangers to the environment.



That's Amore

A Lifelong Affair With Italy

Basin in the sunlight of an Italian dawn, photographer **William Albert Allard** takes to the Po River on the clam boat of Mario Moretti. "It's beautiful being out there," says Bill, "but clamming is one

of the most backbreaking jobs you can imagine." Bill first worked in Italy in 1969 for a book on the Renaissance, returned to teach a photographic workshop in Tuscany, then made pictures for our 1995 article on Sicily.

Though he's worked in some 25 countries, and recently published a retrospective of his work at home—*Portraits of America*, Bill admits he's "always looking for an assignment in Italy. I love Italians; they love life."

MARISA MONTIBELLER—ABOVE; AGHAN DANIEL

PERU

Not Even a Trim?

In Peru to document the excavation of newly discovered mummies, **Ira Block** took time out to photograph other activities in the village of Tupac Amaru and wandered into a barber shop (right), where he was teasingly told he needed a haircut. He declined the offer. "I'd gotten a haircut just before going to Peru," he says.

Ira made the second of his two trips to Peru in late September. Leaving his home in New York City just weeks after the attacks that destroyed the World Trade Center, he reverted to old habits: He took a shortwave radio with him. "In recent years everywhere



DANA ROMANOFF

you go has CNN," he says, "but this time I was a little more concerned and knew I might not be in a place with TV." Ira found the

local residents sympathetic to events in the U.S. "They'd lived with terrorism for ten years in that part of Peru," he says.

WORLDWIDE

For her first assignment for the magazine, photographer **Nina Berman** (below) shot this month's ZipUSA on Chicago's Lincoln Park. It seems natural: She lived in Chicago for six years. But Lincoln Park is on the North Side of the city, while Nina had lived on the South Side, and ■ any Chicagoan can testify, rarely do the twain meet. "I knew the city, though, and I'm a city person," she says. The assignment had other challenges. "I spent a lot of time looking for apartments that had ■ good view of the train going by," Nina recalls. "The fun part is when you finally discover the place you dream of, and you can start making pictures."



SMITH

Author **Shane DuBow** has known Chicago even longer than Nina. He grew up in the area and, except for a few college years, has lived there all his life. These

days he lives one zip code south of Lincoln Park: "Everyone calls my area Ukrainian Village except for the real estate agents, who call it West Town." Shane says he worked hard to see Lincoln

Park with fresh eyes. "I had to ■ it new, not have a lot of preconceived ideas about what the neighborhood was," he says.

Sometimes working on a story is life altering. That happened to **Jennifer**

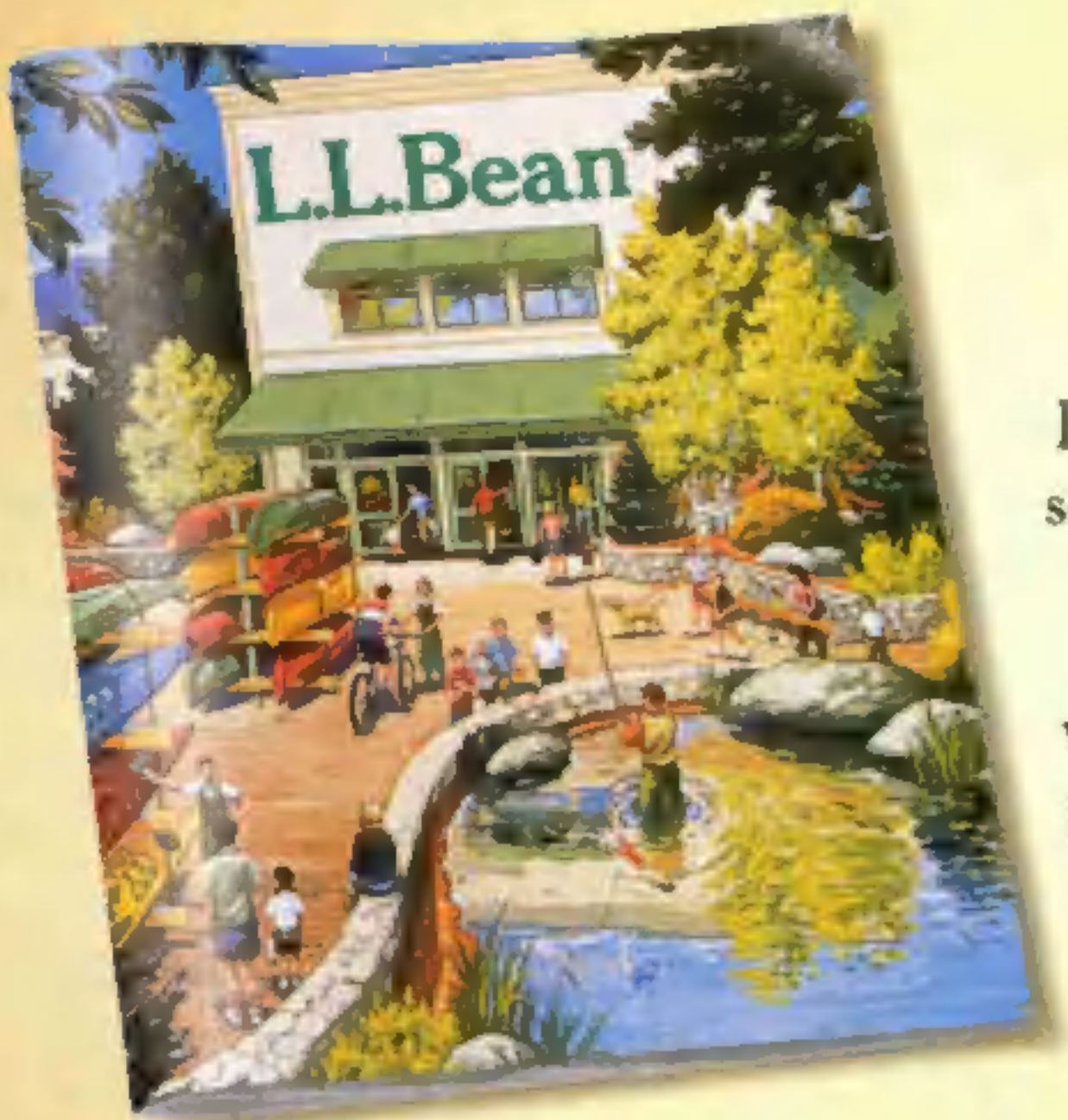
Ackerman ■ she investigated the safety of the nation's food supply. "There was a point at which I rued my new knowledge," she reflects. "It seemed to ruin eating, one of my greatest pleasures in life." Jennifer ended up putting risk in perspective but also made changes ■ how she buys and prepares food. She no longer eats rare hamburgers; she has become a self-described

"fanatic" about washing produce; and she's much more conscious of what she orders in restaurants. Jennifer ■ trying to help her daughters, ages 9 and 6, understand some of the dangers that lurk ■ tainted food. "Sometimes they think I've gone off the deep end, but the problem is real."

In retrospect, author **Fen Montaigne's** trip on a Guyanese river was fun. But while he was there? "The heat and humidity were more enervating than I'd expected, and the number of things that could bite you or do you harm was unbelievable," Fen says. "Poisonous snakes, bullet ants, and spiders on land, sting-rays and electric eels in the water, and tons of chiggers and mosquitoes—I had bite marks on my body for weeks after I got back."

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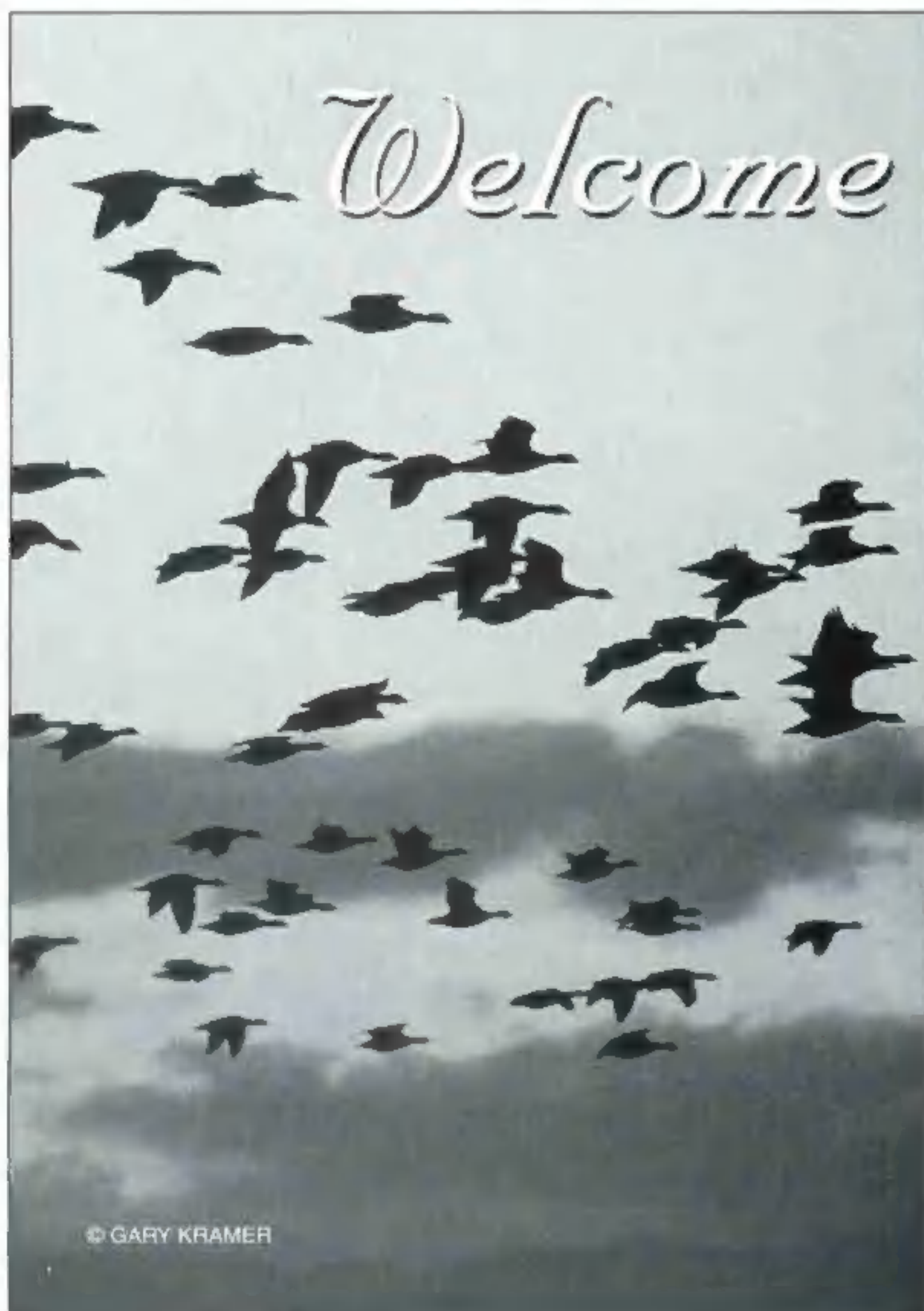
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Welcome

BACK

THE ALEUTIAN *Canada* goose, one of more than a dozen subspecies of Canada geese, is back from a brush with extinction. In the 1960s, there were only a few hundred of these graceful migratory birds left. Today they number more than 30,000, thanks in large part to conservation efforts by Ducks Unlimited and others. That's a job well done, but more work remains. Join us to see how you can help.



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Flashback



J. C. ALLEN & SON

FOOD

Hogs Wild

Pigs carved from lard sing their own praises in a display at the International Livestock Show in Chicago in 1942. First published in an April 1943 story about wartime agriculture, "Farmers Keep Them Eating," the photo underlined a looming concern: slim supplies of fat.

Author Frederick Simpich predicted a "fat famine," citing a call by the United States Department of Agriculture for more fat-supplying foods to fill the demands of World War II troops, lend-lease agricultural aid to allies, and Americans' own appetites. "Day and night rises the cry for more lard and pork, louder and louder," he wrote.

At the time Simpich's story was published the average American ate 52 pounds of animal and vegetable fat a year. Nowadays the annual total is closer to 60 pounds.

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